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THE COMMON COLD*

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The nose contains three different types of epithelium: (1) the squamous epithelium in the vestibule, (2) the respiratory epithelium and (3) the special sense epithelium.

The squamous epithelium of the vestibule is simply a portion of the skin that extends within the nostril. It does not enter into the present study.

The respiratory epithelium takes in the largest portion of the nose. It extends from the squamous epithelium anteriorly to the squamous epithelium of the nasopharynx posteriorly and from the floor of the nose up to the olfactory area. The respiratory epithelium is pseudostratified, ciliated columnar in type (figs. 1 and 2). The superficial layer is composed of long columnar cells, each of which is surmounted by a tuft of rather long cilia. It contains many goblet cells. There are usually from three to five layers of these. All of the epithelial cells, including the surface columnar cells, extend through the basement membrane. The basement membrane is a rather thick, fibrous layer which may be either straight (fig. 1) or festooned (fig. 4), depending on whether or not the underlying erectile tissue is engorged. The basement mucous membrane contains many tiny canaliculi through which lymphocytes and polymorphonuclear cells may travel.

The structure of the basement membrane and its festooned appearance when the blood channels of the erectile tissues are empty suggest the thought that this basement membrane may also be a limiting membrane; that is, it determines the extent of the swelling of the erectile tissues.

The subepithelium consists of a rather loose connective tissue (fig. 1). Just beneath the basement membrane, there is a layer of scattered lymphocytes. The lymphocytes are confined to the more superficial portion next to the epithelium, except in the region of the

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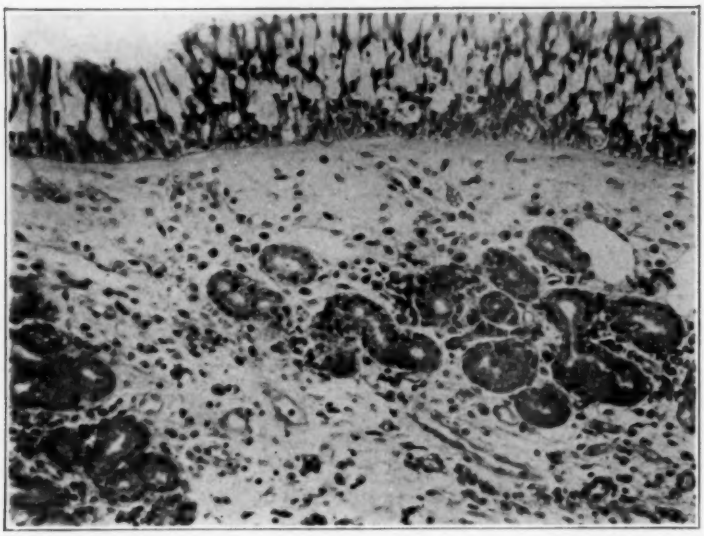

Fig. 1.—Normal nasal epithelium showing many goblet cells. The basement membrane here is fairly well defined. A compound racemose gland surrounded by normal lymphocytes and capillaries is seen in the submucosa.

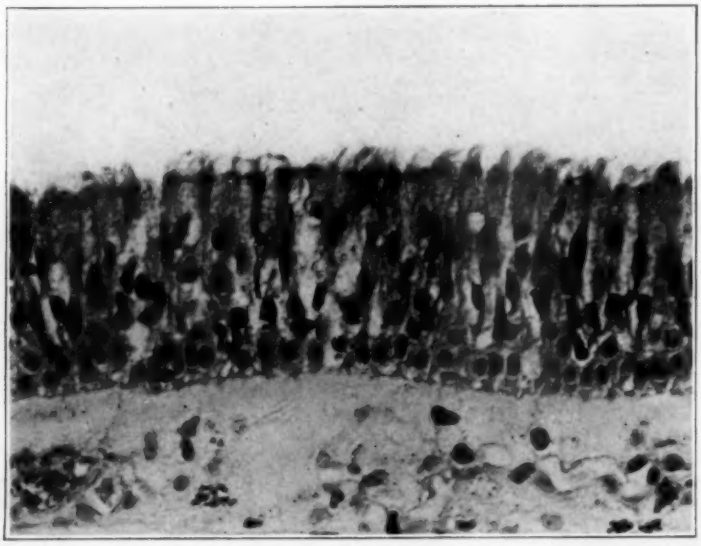

Fig. 2.—Normal nasal epithelium under high power lens, showing the columnar cells surmounted by cilia and the deeper stellate cells next to the basement membrane.

deeper glands. These are surrounded by a scattering of lymphocytes (fig. 1). There are other wandering cells in the subepithelial tissue, prominent among which are eosinophils. Compared to the lymphocytes, these are few.

The glands vary considerably in their size and structure. Some are very simple and others very complex. The simplest are the single goblet cells that occur in the epithelium (fig. 1). These are in effect one cell glands (fig. 11). It is thought that they die after secreting a globule of secretion (Grossman). The next gland in order of complexity is a simple tubular gland which is found in the intra-epithelium. Then follow the simple branching glands extending into the subepithelium, and finally the compound racemose gland (fig. 1). The deeper glands are serous, mucous and mixed.

PRESENT STATUS OF THE COMMON COLD

Can the common cold be conquered and eradicated by medical science as other diseases have been?

This question is of utmost importance for two reasons: first, because of the suffering the loss of efficiency and the loss of life due to the cold itself or to its numerous complications; second, because of the tremendous economic loss through lost time.

According to Blanton's [1] statistical studies on 800 persons, the average was 1.7 colds apiece a year. In the aggregate, 1.7 colds a year, lasting on an average four days each, throughout the entire population represents a tremendous loss of time. It is scarcely necessary to mention the complications: sinusitis, ear infections, mastoiditis and intracranial complications such as meningitis from all of these; also bronchitis, pneumonia and cardiac and renal as well as many other infections. The dangers of these conditions are common knowledge.

It seems strange that such a common and mild disease should not have been conquered by medical science, which has wrought such wonders in other fields. The failure is not due to lack of effort. The literature of the last hundred years shows evidence of much patient and careful work, especially on the etiology. In the great confusion of reports and opinions, a few facts and theories stand out clearly.

Colds seem definitely to fall into two groups: (1) those due to exposure and (2) those due to infection.

The following evidence in my opinion shows that colds do come as a result of exposure:

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1. Blanton, Wyndham, B.: Virginia M. Monthly **53**:787, 1927.

1. The all but universal personal experience that wet feet and exposure to damp, cold weather is followed by a cold.

2. The experiments of Grant, Mudd and Goldman.[2] These workers chilled the surface of the body by placing the feet in cold water and directing electric fans against the bare skin. Then they measured the drop in temperature of the mucous membrane of the nose and throat by means of an electric thermopile. A marked ischemia resulted. The loss of temperature was found to be as great as 6 C. in the nose and from 1 to 2 C. in the nasopharynx. This very striking change in temperature was followed by a reaction and hyperemia. There was no change in the temperature of the blood corresponding to the reduced temperature of the mucous membranes. In several of the volunteers used in these experiments sore throat or colds developed.

3. The statistical studies of Gahwyler [3] during the World War. He did two experiments involving 8000 men; 2,700 were sent out into the cold, wet trenches for three days and three nights, and the remaining 5,300 were kept in barracks. The incidence of colds in the 2,700 exposed was four times as great as in the controls. Later, 4,500 men were sent out for three days in a sharp wind; 3,500 were kept in their quarters. The incidence of colds among those exposed was four times that of those who remained in their barracks.

4. Animal experiments. There are numerous animal experiments which show that chilling greatly increases susceptibility to infections (Lipari, Fischl and Lode [4]). It may be that exposure plays no further rôle than increasing susceptibility to infection.

The following facts point toward infection:

1. The almost universal experience of developing a cold after close association with some one else who has a cold.

2. The tendency for colds to attack progressively many members in one family.

3. Inoculation experiments of Foster,[5] Kruse,[6] Robertson and Groves [7] and others who have produced colds by spraying filtered secre-

2. Grant, S. B.; Mudd, S., and Goldman, A.: J. Exper. Med. **32**:87, 1920.

3. Gahwyler, M.: Schweiz. med. Wchnschr. **52**:648, 1922.

4. Lipari; Fischl, and Lode, quoted by Gahwyler (footnote 3).

5. Foster, G. B.: Etiology of Common Colds, J. Infect. Dis. **21**:451 (Nov.) 1917.

6. Kruse, W.: München. med. Wchnschr. **61**:1547, 1914.

7. Robertson, R. G., and Groves, R. L.: Experimental Human Inoculations with Filtered Nasal Secretions from Acute Coryza, J. Infect. Dis. **34**:400 (April) 1924.

tion from cases of colds on the nasal mucous membranes of volunteers. Other experimenters have failed to produce colds in this way.

4. Reports of small epidemics in groups of closely associated persons (Larrabee [8]).

5. Studies of the relation of colds in infants to colds in the mothers. Winholt [9] studied 500 infants with colds, controlled by 500 without colds. More than twice as many mothers in the first group had colds as in the second.

None of these points taken alone proves that colds are caused by infection, but taken together they are highly suggestive.

A tremendous amount of work has been done on the bacteriology of colds. Some of the best work of this nature was done by the following bacteriologists: Williams, Nevin and Gurley,[10] who grouped the nasal flora into four groups and flora from year to year; Ivens and Stern,[11] who studied the flora in infants with colds; C. H. Benham,[12] who compared the bacterial flora from year to year. There have been many other investigators. The most striking fact brought out in these studies is that the predominating organism varies between years, epidemics and individuals. It is not constant, as would be expected in a disease entity. The reasonable conclusion seems to be that the common cold may be caused by any one of many different bacteria, and that the symptom syndrome termed "a cold" is simply the nonspecific reaction on the part of the nasal mucous membrane to any infection. Conjunctivitis may be caused by any one of half a dozen different bacteria: diphtheria, streptococcus, Koch-Weeks, pneumococcus, xerosis or the diplobacillus. Meningitis, peritonitis and pneumonia are also definitely caused by various infections. Is it reasonable to assign infections of the much exposed nasal mucous membranes to a single organism or a group of organisms?

Clinically, colds differ materially. Some epidemics are accompanied by fever; others are not. Some are characterized by great prostration, others by ear complications, others by laryngitis and still others by pneumonia or rheumatic pains.

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8. Larrabee, R. C.: Boston M. & S. J. **158**:191, 1908.

9. Winholt, W. F., and Jordan, E. O.: Epidemiology of Colds in Infants: Studies on Respiratory Division, J. A. M. A. **81**:280 (July 28) 1923.

10. Williams, Anna W.; Nevin, Mary, and Gurley, Caroline R.: J. Immunol. **6**:5, 1921.

11. Ivens, Paul, and Stern, G.: Jahrb. f. Kinderh. **45**:165, 1921.

12. Benham, C. H.: Brit. M. J. **2**:1338, 1909.

The bacteriology of complicating lesions bears out the theory of multiple causes, because the organisms found in sinusitis, otitis media and other complications differ in different persons and during different seasons.

Considering the evidence for these two groups of colds, exposure and infection, it seems probable that the two may be combined. That is, infection may secondarily invade the mucous membranes, the resistance of which has been reduced by the ischemia and lowered temperature. If that is true, then exposure is merely a contributing factor, and one is back to a very old theory; namely, that colds were caused by reducing the body resistance through chilling and thus rendering it susceptible to infection by bacteria.

If, then, colds are merely the manifestation of a nonspecific inflammation of the nasal mucous membrane, obviously the finding of any specific or adequate treatment becomes highly improbable, if not impossible. The experience of the medical profession to date seems to show this to be true. This would account also for the difficulty and uncertainty of the experimental reproduction of "colds" in volunteers and animals. For instance, a person peculiarly susceptible to a certain mildly pathogenic strain of diphtheroid bacillus develops a "cold" caused by that organism. Attempts to transmit such a cold to volunteers would in all likelihood fail, because very few would be susceptible to that strain.

PATHOLOGY OF THE MUCOSA DURING COLDS

For over two years I have attempted to learn something of the nature of colds by studying the secretion and the mucous membrane microscopically and by various experiments. The secretion has been examined at different stages in both fresh and stained smears, and the mucous membrane has been studied by means of about 100 scrapings and 25 biopsies.

Biopsies.—The twenty-five biopsy specimens were removed from the anterior third of the inferior conchus at different stages, varying from the second hour to the second week of attacks of "colds." The removal of a specimen is not easy. The epithelium is so delicate that it is difficult to remove a piece without crushing or scuffing the surface. For some reason the removal of a tiny bit of mucous membrane during a cold is followed by profuse and stubborn bleeding. It is usually necessary to leave a pack firmly in contact with the spot for twenty-four hours. Even then it may bleed badly when the pack is removed. On account of this bleeding, one hesitates to take biopsy specimens.

The results of the biopsies are briefly indicated in table 1.

The pathologic process of a cold as shown in these twenty-five tissues is about as follows: When the first irritation begins, there is an edema in the submucous membrane (fig. 3). This is followed by an infiltration of polyblasts with a few polymorphonuclear cells (figs. 4 and 6). The first change seen in the epithelium is the separation of the cells as though a fluid formed between them. They loosen, and the surface cells begin to slough off (figs. 4 and 5). A necrotic zone may

TABLE 1.—*Pathologic Process in Twenty-Six Biopsy Specimens*

Subject	Time Taken	Edema	Pathologic Cellular Infiltration	Surface Sloughing*	Surface Regeneration	Bacteria in Tissue
H. A.	Normal	0	0	0	0	0
D. T.	Normal	0	0	0	0	0
J. H.	Normal	0	0	0	0	0
H. A.	2d hour	±	+	0	0	0
N.	1st day	0	+++	?	0	
A. O.	2d day	Specimen badly crushed				
M. E.	2d day	..	+	Specimen crushed		
C. M.	2d day	..	+	+	0	
D. T.	2d day late	0	++	++	0	0
A. D.	3d day	0	++	+	++	0
H. A.	3d day late	0	++	+	?	0
M.	3d day	0	+++	+	++	0
G.	3d day	0	+	+	0	0
M. E.	3d day	0	+++	++	0	0
B. N.	4th day	0	++	+	+	0
McK.	4th day	0	+++	++	?	0
P.	5th day	+?	++	+++	+	0
B.	6th day	0	+++	++	++	0
A.	6th day	0	+	+?	++	0
R. K.	8th day	0	++	?	++	
N. A.	10th day	0	+++	+	+++	0
K. G.	14th day	0	+	++	++	0
H. G.	14th day	+	+	?	++	0
F.	Chronic sinusitis	+	+	?	?	0
L.	Chronic sinusitis	+	+	?	?	0

* The degree of surface sloughing was sometimes uncertain because of mechanical scuffing in taking the specimen.

form next to the surface (fig. 7), or single cells or groups of cells may show necrosis (fig. 12). This necrosis may extend deep into the mucosa (fig. 8). Then almost the entire epithelium disintegrates; it looks as though it had been exploded. Many, and sometimes all, of the columnar cells are gone by the third day (fig. 9). Some of the stellate cells close to the basement membrane also slough out (fig. 10). The nasal secretion contains many epithelial cells during the first two or three days (fig. 13). Sometimes there may be only a single layer left. The remaining cells proliferate after the most acute stage has passed, until they are many layers deep (fig. 14). A syncitium forms at the

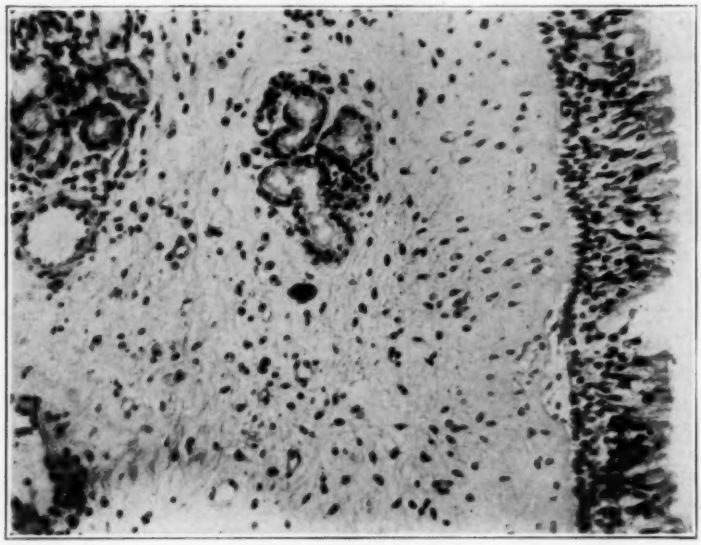

Fig. 3.—Edema of the submucosa in the second hour of a cold.

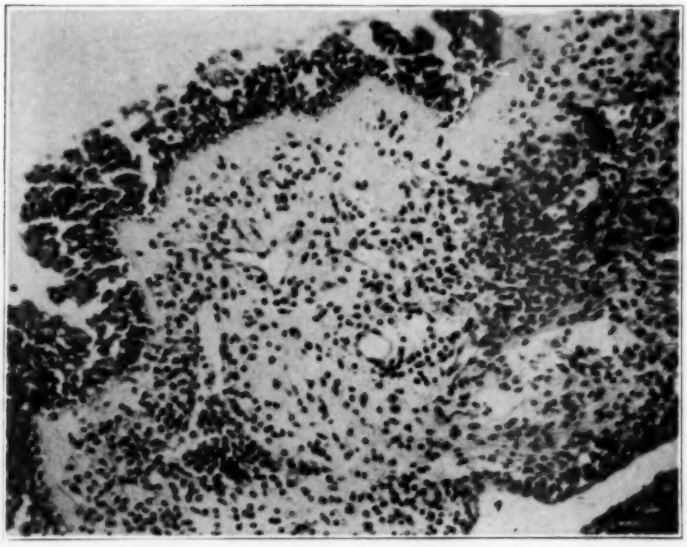

Fig. 4.—Epithelium and submucosa during the third day of a cold. The epithelium is disintegrating and the submucosa shows a proliferative inflammation.

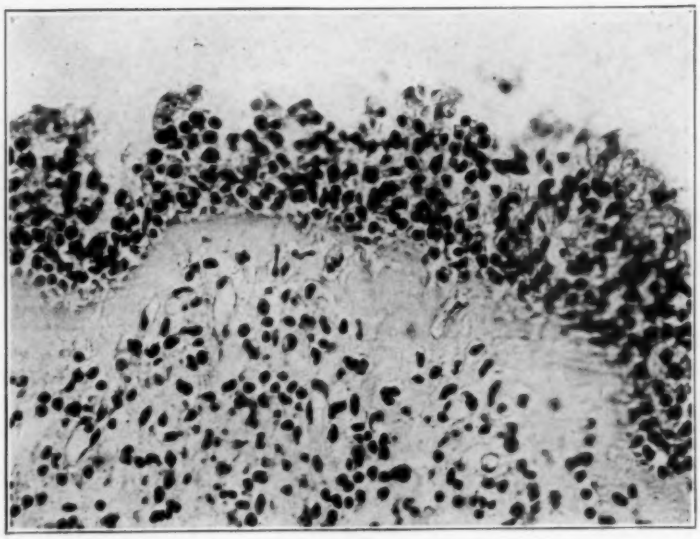

Fig. 5.—High power view of the disintegrating mucosa. Same specimen as figure 4.

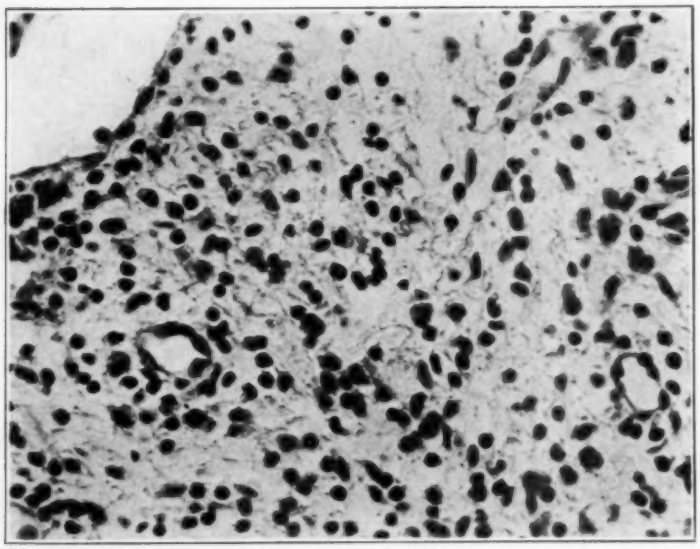

Fig. 6.—Character of the polyblastic infiltration in the submucosa. There are a number of eosinophils in this field. Same specimen as figure 4.

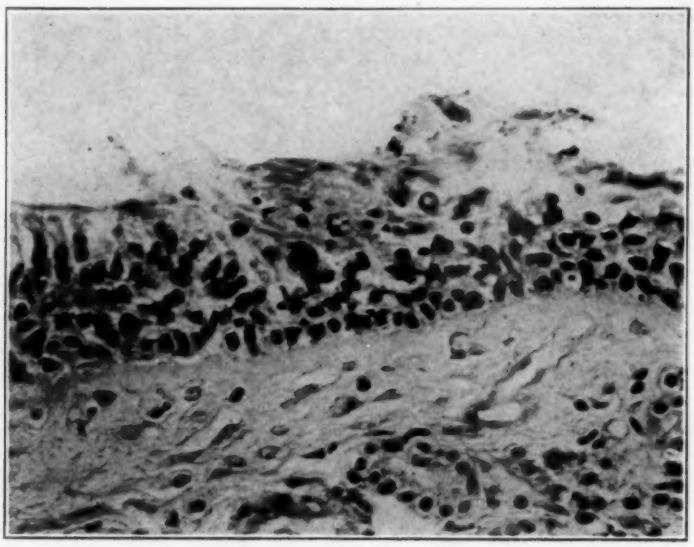

Fig. 7.—Epithelium showing necrosis of the superficial layers on the third day of a cold. The necrotic mass is still in place and shows cell shadows.

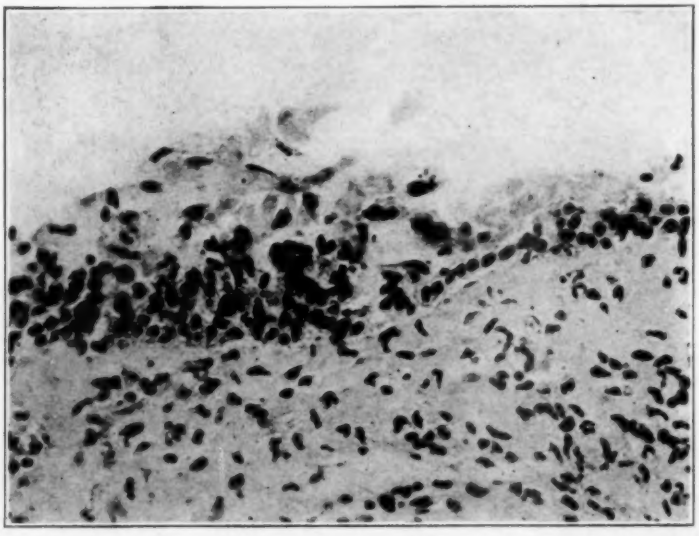

Fig. 8.—Disintegration of the epithelium with necrosis extending to the last layer of cells, on the fifth day of a cold.

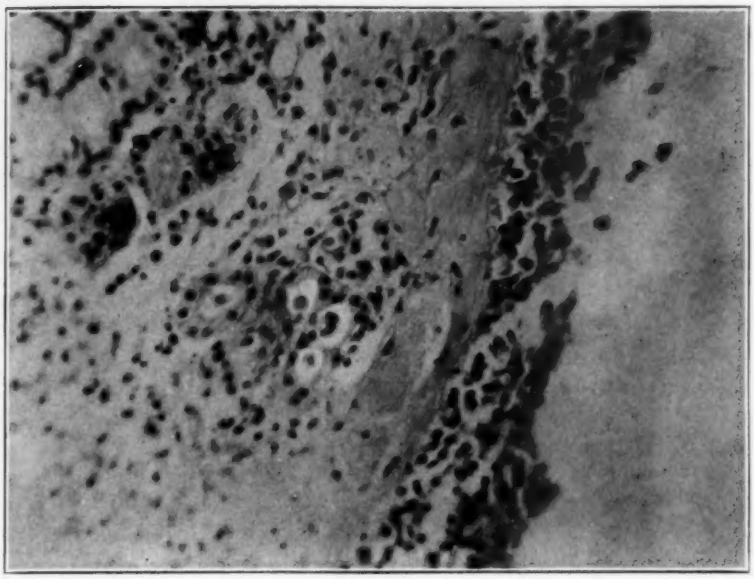

Fig. 9.—Disintegrating mucosa showing surface cells largely sloughed away.

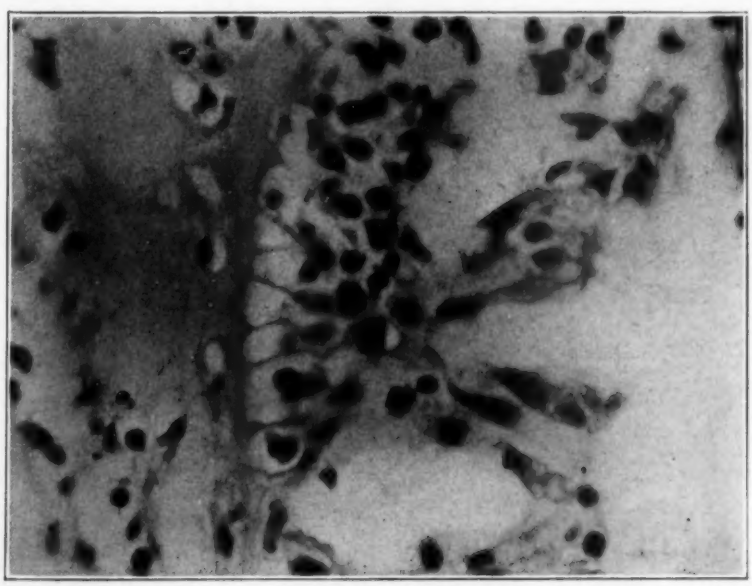

Fig. 10.—High power view showing detail of loosening epithelium on the third day of a cold.

Fig. 11.—Active epithelial cell found in a scraping taken the second day of a cold.

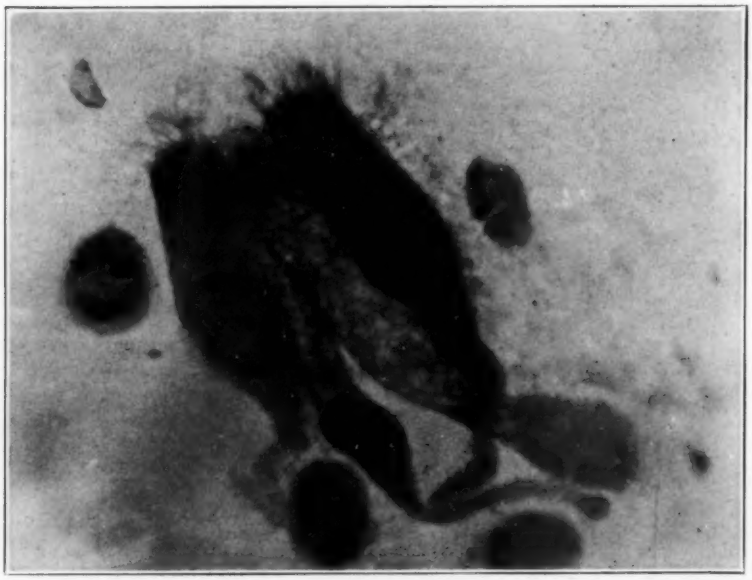

Fig. 12.—Epithelial cells and polymorphonuclears removed by scraping on the second day of a cold. Note the faint staining of one of the epithelial cells. This shows necrosis.

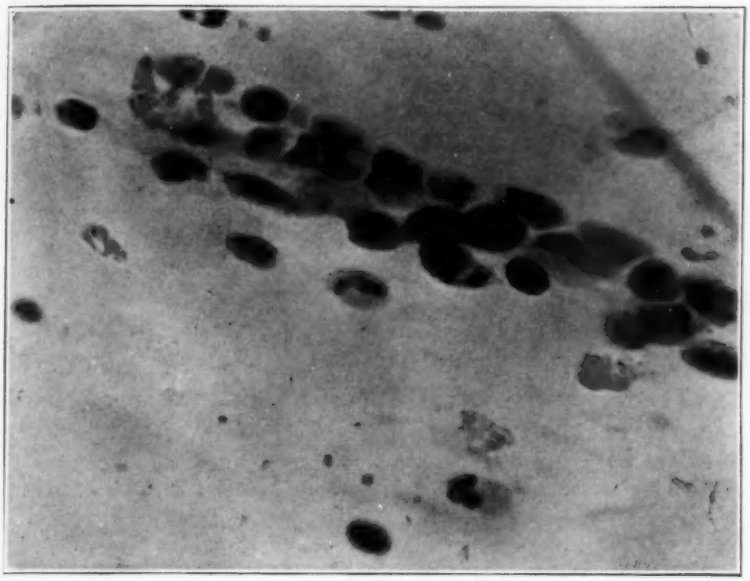

Fig. 13.—Epithelial cells and polymorphonuclears in the secretion on the third day of a cold. The cilia do not show readily in the stained smears and the columnar cells have become rounded. In the fresh warm secretion some of the epithelial fragments move rhythmically by ciliary action.

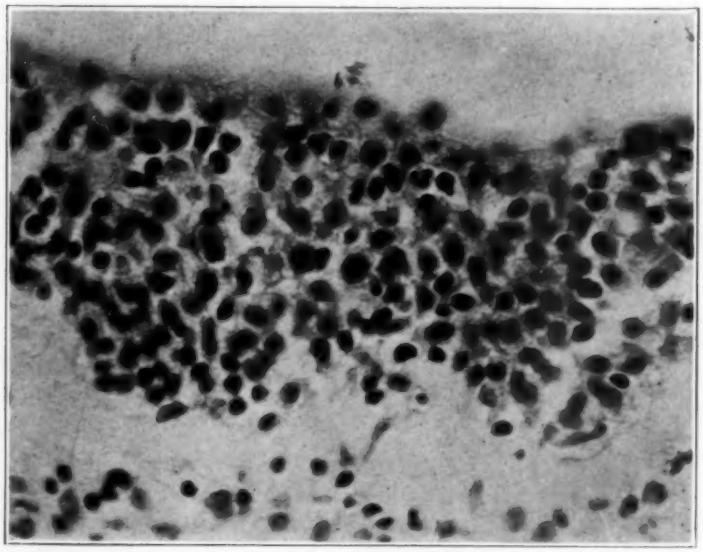

Fig. 14.—Proliferating epithelium on the eighth day of a cold. Many pus cells are seen between the epithelial cells. The surface layer is intact after repair.

surface (figs. 14 and 15). Finally, the surface cells begin to lengthen until they again look like columnar cells (fig. 16).

The inflammatory reaction of the submucosa is of the proliferative type (fig. 15). The cellular infiltration is largely composed of variously shaped mononuclear cells with a scattering of polymorphonuclears and some eosinophils (fig. 6). The edema soon decreases, but the cellular infiltrate increases. By the second or third day, many polymorphonuclear cells and polyblasts are found in the epithelium on their way to the surface (fig. 14). They increase in number as the purulent stage develops.

Repair seems to take place even while there is still much secretion from the nose. Figure 14 shows the condition on the eighth day of an active cold with a many-layered epithelium full of polymorphonuclears. The surface is again intact. Figures 15 and 16 were taken on the tenth and fourteenth days, respectively. There was a profuse nasal secretion in both cases. The epithelium in both is well regenerated, and in figure 16 the cells are taking on a columnar form.

Bacterial Examination.—Many serial sections of the mucosa and submucosa in all the different stages were examined for bacteria with several different stains (polychrome methylene blue, Gram-Weigert, iron hematoxylin). None was ever found within the mucosa, although there were many on the surface in the secretion. This is a striking fact. The bacteria seem to stay on the surface as in diphtheria and do not penetrate between the cells. It may be, of course, that further search will reveal them among the cells. (Further study on this point has been begun.)

Secretion.—The cellular content of the secretion changes rapidly. The watery secretion of the first few hours contains comparatively few cells. Sometimes within the first twenty-four hours living ciliated epithelial cells or fragments of them appear in the secretion. They move with a rotary, rhythmic movement usually through a half circle. The rate varies from 60 to 268 beats per minute. They become more numerous during the second day and usually disappear after the third (table 2). They may disappear very suddenly . For instance, L. E. A. had swarms of them in the forenoon of the third day. By later afternoon there was none. They are most numerous when the secretion is clear and disappear when the secretion is purulent. They assume many different sizes and bizarre shapes (fig. 17). At first glance some of these might be mistaken for parasites. The deeper epithelial cells, polyblasts and polymorphonuclear cells appear in the secretion on the second day and rapidly become more numerous. The polymorphonuclears, especially, increase until after a few days these make up most of the cellular content of the secretion.

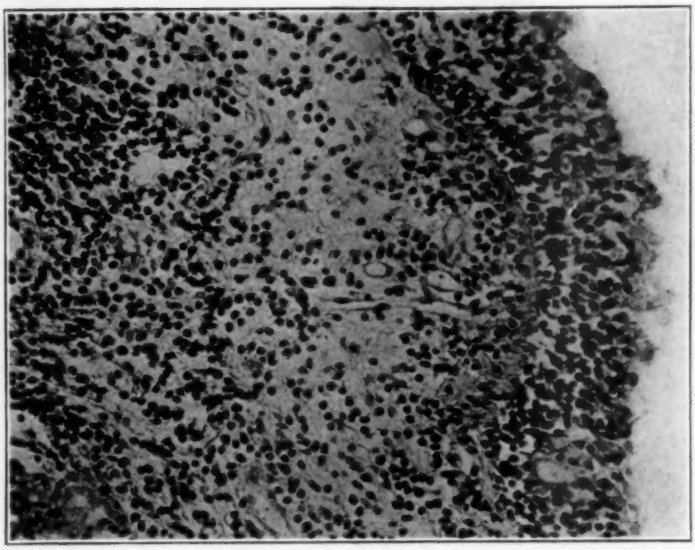

Fig. 15.—Appearance of the mucosa and submucosa on the tenth day. The mucosa is proliferating.

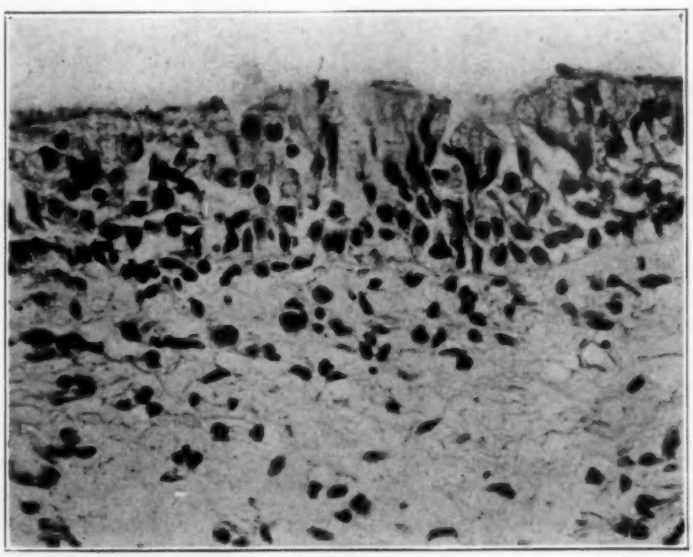

Fig. 16.—The epithelium is approaching normal appearance. Some columnar cells are developing. There was still some nasal discharge. This specimen was taken on the fourteenth day.

TABLE 2.—*Days on Which Living Fragments of Ciliated Epithelial Cells Were Found in the Secretion*

	First	Second	Third	Fourth	Fifth	Sixth	Seventh	Eighth
M. R.	..	..	+++	+	..	..	..	..
M. W.	..	..	..	+	..	..	..	..
E. L.	..	..	0	..	..	..	..	..
N.	..	++	..	..	..	..	..	..
J. B.	0	..	..	..	..	..	..	..
J. H.	..	..	..	..	0	..	..	..
L. A.	..	..	..	0	..	..	..	..
K.	..	..	0	..	..	..	..	..
S.	..	..	0	..	..	..	..	..
J. H.	++	++	+	0	..	..	..	..
H. A.	0	++	+	0	..	..	..	..
W.	++	+	+	0	..	..	..	..
M. E.	..	..	+	..	..	..	..	..
L. A.	..	+++	..	..	..	..	..	..
M. E.	+	++	..	..	..	..	..	..
M. A.	..	..	..	..	..	..	0	..
M. S.	..	..	..	..	..	..	0	..
M. G.	..	+++	++	+	..	..	..	..
B.	..	..	..	0	..	..	..	..
M. E.	..	+	..	..	..	..	..	..
M. E.	+	+	..	..	..	..	..	..
S.	..	+++	..	..	..	..	..	..
L. E. A.	..	..	+++	..	..	..	..	..

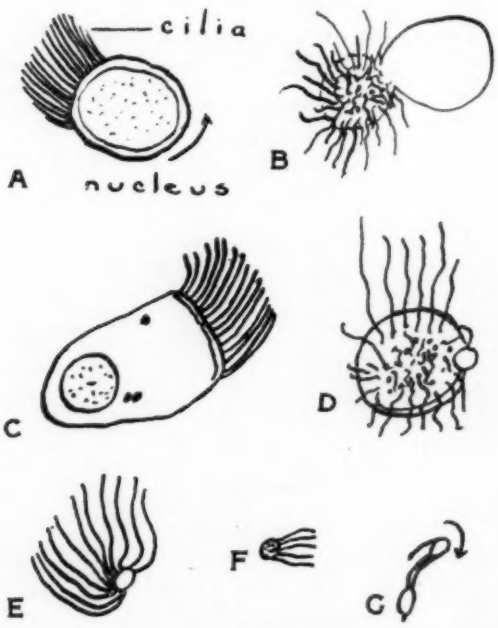

Fig. 17.—Fragments of ciliated epithelial cells found in the fresh nasal secretion during the first three days of a cold. The swinging rhythmic motion is due to the whiplike action of the cilia. In *A* the rhythmic movement varied from 63 to 67 beats per minute; *B* shows the cilia as seen from above; *C*, the common form divided just proximal to the nucleus; *D*, a fragment that moved with a pulsating movement in a hanging drop for two and a half hours, and *E*, *F* and *G*, very small fragments with marked rhythmic movements.

It has often been stated that acidosis obtains in a cold, and that the secretions are more acid than normally. To test the truth of this claim, I determined the hydrogen ion concentration in seventy different secretions, and found the opposite to be true. The p_H rose slightly, i. e., became more alkaline in the first four days of a cold and then fell slightly below normal in the purulent stage. But it always remained alkaline. Naturally, this proves nothing concerning the alkaline reserve of the blood. These slight changes in the p_H of the nasal secretion are not enough in the opinion of Dr. W. P. Larson of the Department of

TABLE 3.—*P_H of Secretion at Different Stages of Colds*

Normals	First Day	Second Day	Third Day	Fourth Day	Fifth Day	Sixth Day	Seventh Day	More Than 7 Days
8.12	8.4	8.2	8.3	7.9	7.1	...	7.7	8.4
8.2	8.1	8.1	8.25	8.0	8.9	...	8.1	7.7
8.05	8.5	8.05	8.2	8.2	...	...	8.1	8.1
8.13	8.3	8.3	8.2	8.3	...	...	...	7.7
8.1	8.3	8.38	8.2	8.6	...	...	...	7.6
8.3	8.0	8.4	8.1	8.4	...	...	...	7.2
8.1	8.5	8.2	8.2	...	...	...	...	7.7
8.05	...	...	...	...	...	...	...	8.1
8.05	...	...	...	...	...	...	...	8.4
8.15	...	...	...	...	...	...	...	8.1
8.13	...	...	...	...	...	...	...	8.1
8.1	...	...	...	...	...	...	...	...
7.8	...	...	...	...	...	...	...	...
7.2	...	...	...	...	...	...	...	8.1
7.2	...	...	...	...	...	...	...	7.9
7.2	...	...	...	...	...	...	...	...
7.2	...	...	...	...	...	...	...	...
7.2	...	...	...	...	...	...	...	...
7.6	...	...	...	...	...	...	...	...
7.5	...	...	...	...	...	...	...	...
7.8	...	...	...	...	...	...	...	...
8.4	...	...	...	...	...	...	...	...
Av. 7.9	8.3	8.2	8.2	8.3	(7.5)	...	8.0	8.0

Bacteriology, University of Minnesota, to change the bacterial flora materially (table 3).

Dr. E. Lindner of Vienna has developed a method of scraping the conjunctival surface in cases of conjunctivitis and studying the scrapings for bacteria. He has demonstrated the way in which bacteria invade and form colonies on the cells in the squamous epithelium. This work inspired me to attempt similar studies on scrapings from nasal mucosa taken during colds. About seventy such preparations were made and carefully studied. The columnar cells and some of the stellate cells were easily removed by scraping the mucosa of the septum or medial surface of the inferior concha by means of a small curet. It was necessary to apply cocaine before doing this. The study of my scrapings was negative as far as bacterial invasion of cells was concerned. A great deal of time was spent in staining and examining them for bacteria. Many bacteria were found in the secretion but not a single colony was

ever found on a cell or group of cells removed from the nasal mucosa. (This work is to be pursued further by different staining methods.) The study was principally valuable for information concerning cell forms (fig. 12).

SUMMARY

The common cold does not seem to be a disease entity. According to etiology, it falls into two groups, colds caused by exposure and colds caused by infection. These may be interrelated. The many excellent bacteriologic studies lead one to the belief that many different organisms may cause colds. For this reason, specific treatment and immunization are ineffectual. The pathologic process is that of a mucous membrane inflammation showing rather marked tissue changes, including the loss of many of the surface cells and a proliferative reaction in the submucosa.

The epithelium is regenerated and repaired by the growth and multiplication of the stellate cells normally found deep in the epithelium.

EMBRYONIC TISSUE IN THE MIDDLE EAR AND MASTOID

REPORT OF TWO CASES *

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AND
L. M. POLVOGT, M.D.
BALTIMORE

The following report is interesting and unusual from both a clinical and a pathologic point of view, since it demonstrates the persistence of mesenchyme in the middle ear and mastoid antrum in two persons over 50 years of age. That the tympanic membrane was inspected and an audiometer test was made a short time before death add to the interest of these cases.

REPORT OF CASES

CASE 1.—A white man, aged 63, admitted to the Bay View Division of the Baltimore City Hospital on Oct. 20, 1928, had myocardial insufficiency. He died four days later. The audiometer record (fig. 1) showed an impaired perception for both low and high tones, more marked on the right. The arrow at 8192 indicates that this tone was tested and could not be heard in either ear at the maximum intensity of the audiometer. The tympanic membrane was intact and showed no evidence of infection on either side. The patient's condition was such that tuning fork tests were unreliable. Voice tests, however, with the opposite ear masked, indicated marked impairment: right, ad concham: left, less than 1 meter. There was a definite statement in his records that he had never had an infection of the ear. Unfortunately, we have no record concerning the age at which the impairment indicated in the audiometer chart first became apparent. It would add greatly to the interest of this report if we knew definitely that the patient had been hard of hearing since infancy.

The patient was obese and dyspneic, with a tendency to Cheyne-Stokes respiration, and was very drowsy. The blood pressure was 180 systolic and 110 diastolic. The Wassermann test gave negative results for the blood and spinal fluid. The teeth were in bad condition. The head was deviated toward the right. The examination of the eyegrounds showed marked sclerotic changes in the blood vessels. At autopsy the discovery of a thrombus in the posterior inferior cerebellar artery on the right probably explained the spontaneous vertical and horizontal nystagmus, which was a prominent clinical symptom.

Both the voice test and the audiogram indicated that the hearing was more acute on the left side. The articulation of the malleus and incus and a portion of the mastoid antrum on the left are shown in figure 2. This is a horizontal section. Numerous strands of loose areolar tissue extended from the ossicles to the bony walls of the middle ear cavity. These strands were continuous with the mesenchyme seen in the mastoid antrum. A horizontal section at approximately the

* Submitted for publication, April 10, 1930.

* From the Otological Research Laboratory of the Johns Hopkins University.

same level on the right side showed the articulation of the malleus and incus embedded in mesenchyme (fig. 3). The perception for low tones was more impaired on this side. The stapediovestibular articulation on each side was relatively free (figs. 4 and 5); the niche of the round window, however, was filled with mesenchyme (figs. 6 and 7). Possibly the absorption of embryonic tissue was more advanced on the right (fig. 7).

There is a great variation in the age at which absorption of the mesenchyme in the middle ear takes place. At birth the middle ear is usually filled with embryonic tissue. At varying periods after birth cysts begin to form near the margin and gradually coalesce. The central area is absorbed, and the tissue remaining around the margins becomes the mucous membrane of the middle ear. This process is shown clearly in figure 8, which is a section through the middle ear of a colored girl,

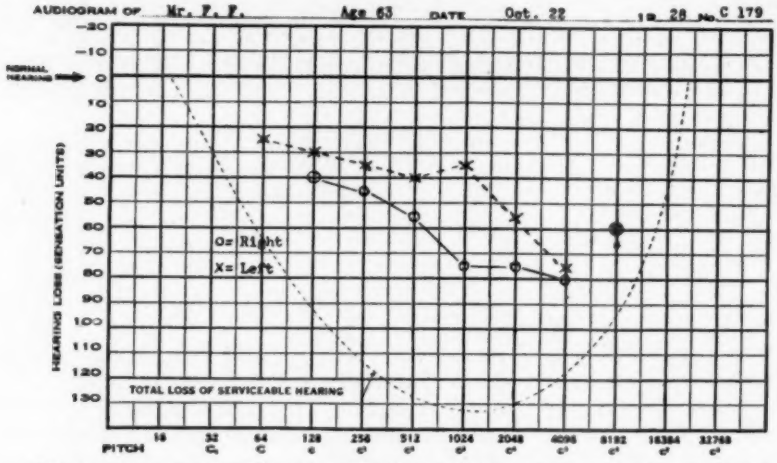

Fig. 1.—The impairment for both high and low tones was more marked on the right; 8,192 double vibrations was not heard on either side.

aged 5 months. Figure 9 shows the appearance of this tissue at a higher magnification. When compared with figure 10, which is an enlargement of the area indicated in figure 3, it is seen that the tissue filling the mastoid antrum in the patient in case 1 is identical with that in the middle ear of a child aged 5 months. We have made no statistical analysis of the material in our laboratory (which consists of serial sections of the temporal bones from more than 1,000 hospital cases) in order to determine the frequency and the various ages of the patients with mesenchyme in the middle ear or mastoid region. We can state definitely, however, that it is not infrequently found in adults. Figure 11, for example, shows mesenchyme in the process of absorption in the niche of the round window and strands of the same tissue around the stapes in a man aged 53 (case 2).

Fig. 2.—Horizontal section through the upper portion of the middle ear and the mastoid antrum on the left. Strands of mesenchyme are seen around the articulation of the malleus and incus and in the mastoid antrum. Reduced from a magnification of 9.5.

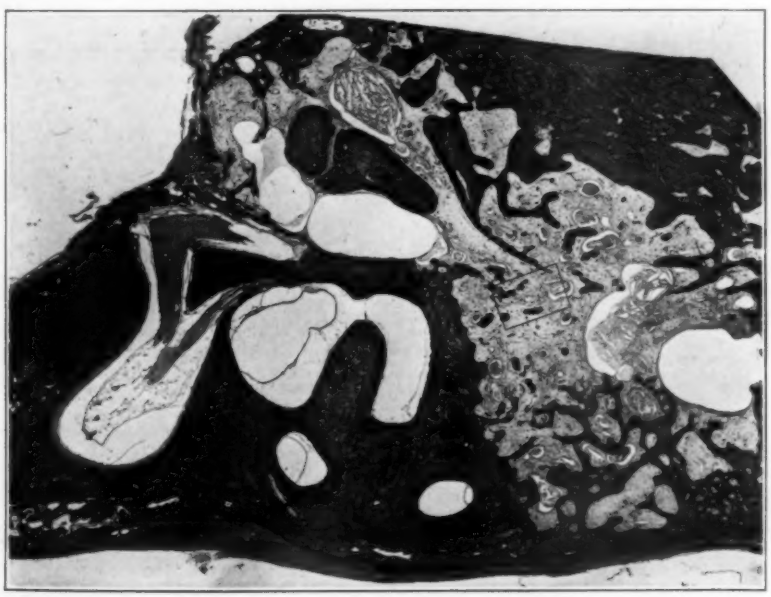

Fig. 3.—Horizontal section through the upper portion of the middle ear and mastoid antrum on the right. Note the mesenchyme around the malleus and incus. Several cysts are seen containing cholesterol crystals. The appearance of the tissue in the marked area is shown in figure 10. Reduced from a magnification of 9.5.

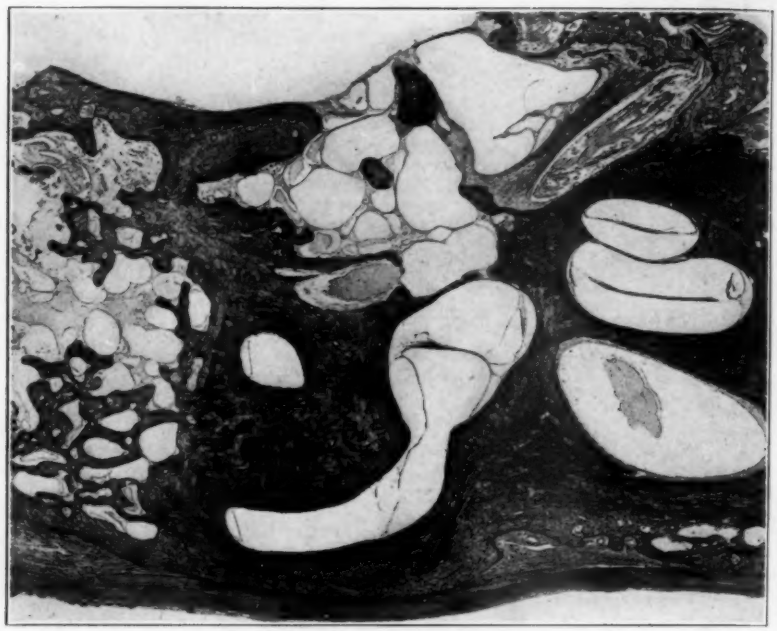

Fig. 4.—Horizontal section through the middle ear on the left to show portions of the malleus, incus and stapes. Note the strands of mesenchyme in the middle ear and mastoid. Reduced from a magnification of 9.5.

Fig. 5.—Horizontal section through the middle ear on the right to show the mesenchyme in the mastoid cells and to a less extent in the cavity of the middle ear. Reduced from a magnification of 9.5.

Fig. 6.—Horizontal section through the left ear at the level of the umbo to show the normal drum, the normal mucosa of the middle ear and the niche of the round window filled with mesenchyme. Most of the mastoid cells were normal at this level. Reduced from a magnification of 9.5.

Fig. 7.—Horizontal section through the right ear at the level of the umbo to show the normal mucosa of the drum and middle ear. The mesenchyme in the niche of the round window is more cystic than that on the left. The mastoid cells at this level are almost solidly filled with embryonic tissue. Reduced from a magnication of 9.5.

Fig. 8.—Vertical section through the middle ear of a child, aged 5 months; the cysts around the margin are typical of absorbing mesenchyme. Reduced from a magnification of 16. The area marked with a rectangle is shown in figure 9.

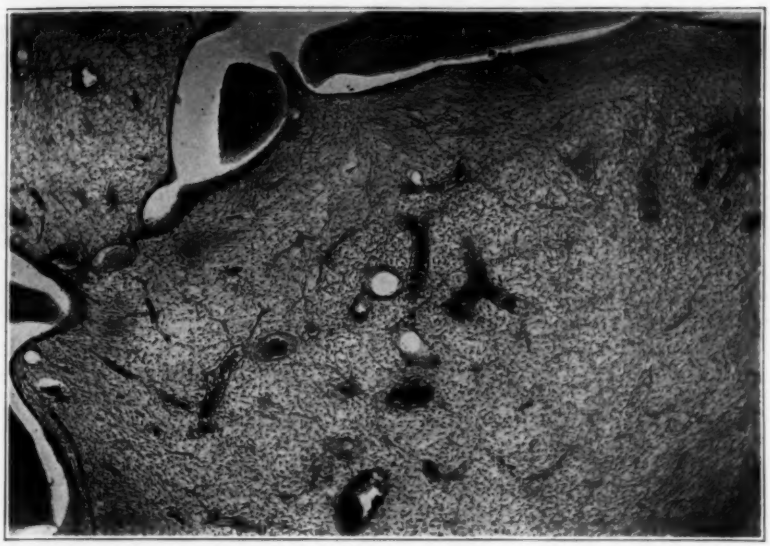

Fig. 9.—An enlargement of the area marked in figure 8. Note the stellate cells, the abundant blood supply and the cysts lined with epithelium; × 71.5.

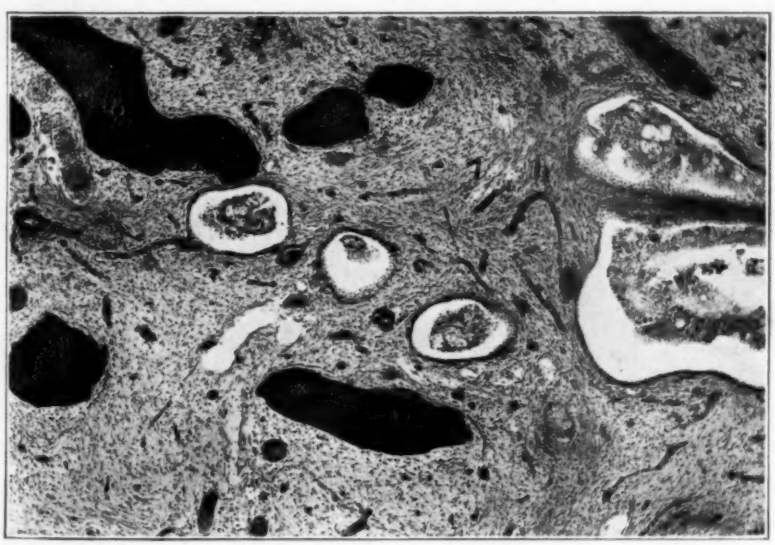

Fig. 10.—An enlargement to show the appearance of the tissue that fills the mastoid antrum in figure 3. This tissue in the mastoid of a man, aged 53, is almost identical with that in the middle ear of a child, aged 5 months (fig. 9). Reduced from a magnification of 71.5.

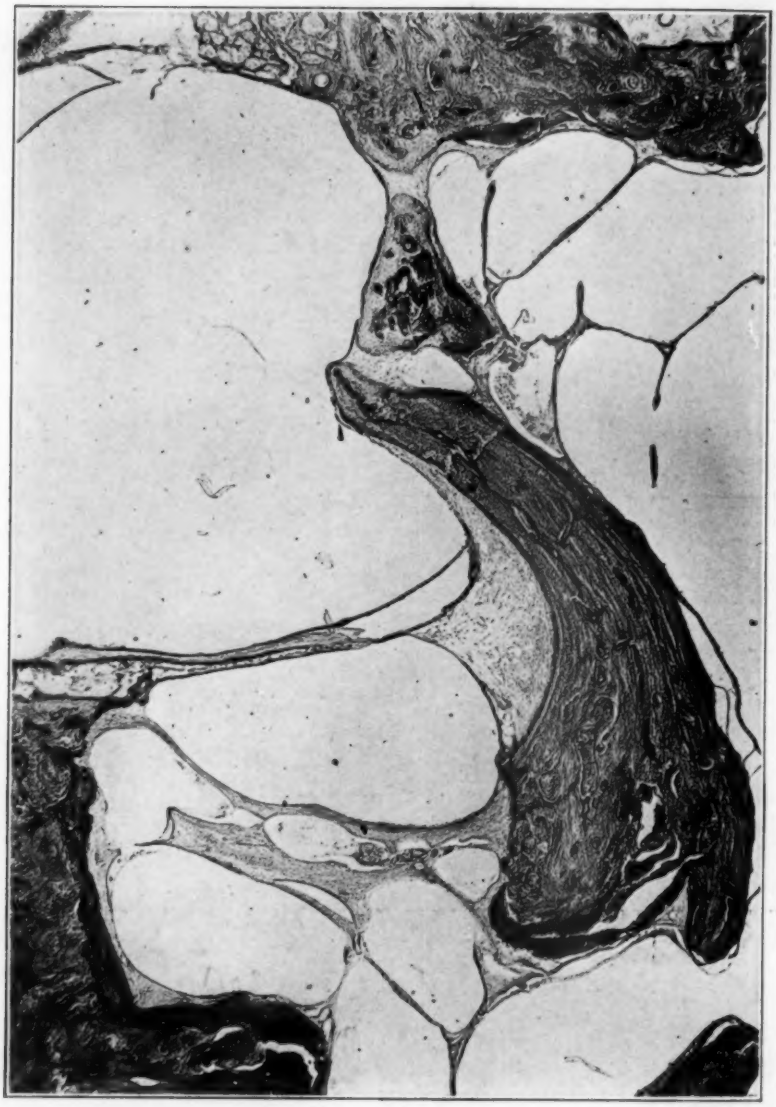

Fig. 11.—Strands of mesenchyme are shown around the stapes and round window. An absence of the organ of Corti in the basal coil of the cochlea, an atrophy of the nerve in the osseous spiral lamina and a calcification of the basilar membrane are also shown. These lesions possibly explain the impaired hearing for both low and high tones, as shown in the audiometer chart (fig. 12). Reduced from a magnification of 29.

CASE 2.—A white man, aged 53, was admitted to the Bay View Divison of the Baltimore City Hospital on June 11, 1928, with an extensive pulmonary tuberculosis. He died on August 20. There was an extensive tuberculous destruction of the tympanic membrane and structures of the middle ear on the right. The drum on the left was normal in appearance. This patient said that he had no trouble with hearing, no infection of the ear at any time and no discharge from the right ear until the present illness. An audiometer chart made on May 31, showed an impairment for all tones, more marked for those above 1,024 double vibrations. The audiometer and the monochord test indicated that this patient could not hear any tone above 10,000 double vibrations by air conduction and 13,000 double vibrations by bone conduction. The audiometer record of this patient is shown in figure 12.

These cases are reported in order to call attention to the fact that embryonic tissue in the middle ear and mastoid may be present in large

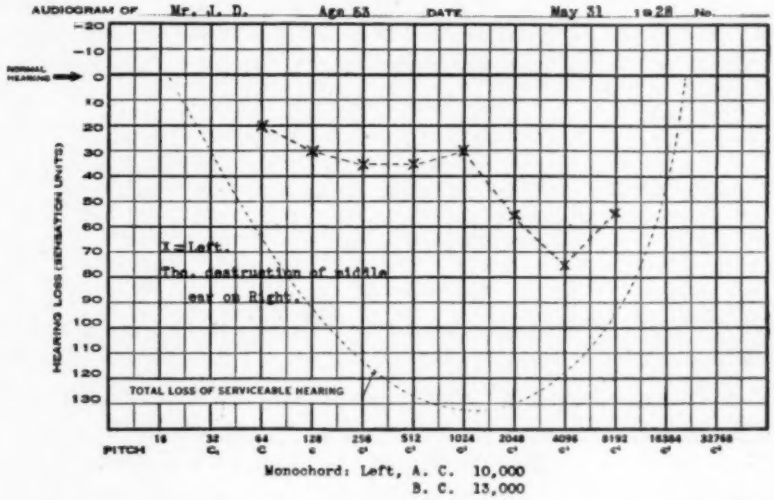
AUDIOGRAM OF Mr. J. D. Age 53 DATE May 31 1928 No.
NORMAL HEARING
HEARING LOSS (RENFREW UNITS)
X = Left.
Tho. destruction of middle ear on Right.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
Monochord: Left, A. C. 10,000
B. C. 13,000

Fig. 12.—Impairment for low and high tones, but most marked for 4,096. The acuity for the right ear is not charted because of extensive tuberculous destruction of the middle ear on this side.

amounts even in late adult life. The audiometer record of case 1 (fig. 1) indicates that there is a lesion of both the middle and the inner ear. This is confirmed by a study of the serial sections. Embryonic tissue is present in the upper half of the middle ear cavity and in the niche of the round window on both sides. The impairment for all tones is more marked on the right, and the malleo-incudal articulation is embedded in mesenchyme on the right. It is impossible to determine in either of these cases how much the mesenchyme in the middle ear and niche of the round window interfered with the hearing. Both patients were quite ill, and the audiometer test was made in an open ward that was fairly noisy. Under other conditions, the perception

for low tones might have been better. Another factor that prevents a clearcut demonstration of the effect on the hearing in these cases of persistent mesenchyme in the middle ear is the lesion of the inner ear, which was present in both cases. It was shown by Bunch [1] that there is an age variation in the acuity of hearing. For each decade after the age of 30 years there is a diminished acuity for high tones. We have so far studied serial sections of the temporal bones of twenty-five patients whose audiometer records, made a short time before death, showed that there was a loss of high tones. In every case there is a correlation between the functional test and the pathologic observations. One or more of the following changes are seen in the basal coil, which would account for the loss of high tone perception: (1) atrophy of the organ of Corti, (2) partial or complete atrophy of the nerve in the osseous spiral lamina, (3) partial or complete disappearance of the spiral ganglion or (4) any combination of the foregoing factors. These facts are mentioned in order to explain the impairment in the perception of high tones in the two cases reported in this paper. Figure 11, for example, which is intended to show embryonic tissue in the niche of the round window and around the stapes, also illustrates the absence of the organ of Corti, the calcification of the basilar membrane and the atrophy of the nerve in the osseous spiral lamina.

CONCLUSIONS

The conclusions drawn from our study of these two cases are as follows:

1. Mesenchyme may persist in certain portions of the middle ear and mastoid throughout life. The niche of the round window, the epitympanum or attic and the mastoid antrum are the usual locations.

2. In such cases the tympanic membrane is normal on clinical examination and in the histologic sections. The presence of abnormal embryonic tissue in the middle ear cannot be recognized from an inspection of the drum. We have not made a roentgenogram of the mastoids in any case, not complicated by infection, that later came to autopsy and proved to have a persistence of embryonic tissue in the middle ear and mastoid. The condition illustrated in figure 3, however, would undoubtedly cause a dense clouding of the mastoid cells and result in a mistaken diagnosis.

3. Histologic study of the temporal bones of the cases reported demonstrates an abnormal condition in the middle ear (a large amount of embryonic tissue) and an atrophy of the nerve and organ of Corti in the inner ear, which is limited, however, to the lower half of the

1. Bunch, C. C.: Age Variations in Auditory Acuity, Arch. Otolaryng. **9**:625 (June) 1929.

basal coil. In this, as in similar cases studied, no definite lesion to a blood vessel could be found which would account for the changes in the nerves and end-organs.

4. Persistent embryonic tissue in the middle ear in adult life is not rare, and in time the perfect combination may appear that will enable us to determine how much, if any, impairment of hearing results from the conditions in the middle ear shown in figures 2 to 8. By the perfect combination we mean: (1) a good clinical note on the appearance of the drums, and history in regard to the hearing and infection of the ear; (2) a satisfactory tuning fork test; (3) an audiometer test made under the most favorable conditions for accuracy; (4) a postmortem examination soon enough after death to insure good histologic results; (5) good fixation, decalcification and embedding; (6) the inner ear normal for the function tests and in the histologic sections; the middle ear uncomplicated by infection.

PNEUMOBACILLUS (FRIEDLANDER'S) MENINGITIS SECONDARY TO OTITIS MEDIA

REPORT OF TWO CASES *

S. MALIS, M.D.
LOS ANGELES

That the pneumobacillus was the cause of many fatal cases of meningitis during the epidemic of 1918 and that many deaths are caused by this organism yearly are well known to the medical profession; yet there appears to be hardly any cases on record in American medical literature. Isolated cases of Friedlander's bacillus septicemia have been described.[1] Brain and Valentine [2] reported a case of infection of the meninges and the blood stream by *Bacillus mucosus-capsulatus* secondary to chronic purulent otitis media of the right ear of almost four years' duration. Two months after the patient had had a mastoidectomy followed by an uninterrupted recovery, he was readmitted to the hospital. He complained of severe cold in the head and that for fourteen days he had suffered from severe headaches and intense pains at the back of the neck. Two days after admission, he died. The organism was isolated from the patient's blood and spinal fluid.

Pneumobacillus septicemias were also reported by Lereboullet and Denoyelle [3] as well as by Caussade, Joltrain and Surmont.[4] Minvielle [5] described a case of suppurative meningitis due to the pneumobacillus secondary to pulmonary infection:

> A man, aged 28, was brought to the Hôpital de la Pitié on Jan. 29, 1924, in a delirious state with a stiff neck and positive Kernig sign. On the following day a lumbar puncture was done, and on the suspicion that the cerebrospinal fluid resembled that in cerebrospinal meningitis, antimeningococcic serum was

* Submitted for publication, Feb. 7, 1930.

* From the Division of Cranial Surgery of Dr. Wells P. Eagleton, Newark Eye and Ear Infirmary, Newark, N. J.

1. Howard, W. T.: The Importance of the Bacillus Mucosus Capsulatus (B. of Friedlander) as the Cause of Acute and Chronic Infections, Philadelphia M. J. **1**:336, 1898.

2. Brain, W. R., and Valentine, F. C. O.: Case of Infection of Meninges and Blood Stream by Bacillus Mucosus Capsulatus, Lancet **1**:855 (April 28) 1928.

3. Lereboullet, P., and Denoyelle: Grave Pneumobacillus Septicemia, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:226 (Feb. 29) 1924.

4. Caussade, G.; Joltrain, E., and Surmont, J.: Pneumobacillus Septicemia, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:148 (Feb. 8) 1924.

5. Lion, G., and Minvielle: Suppurating Meningitis with Friedlander's Bacillus, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:286 (March 7) 1924.

injected. Toward evening, the patient went into coma and died thirty hours after admission to the hospital. Necropsy revealed a suppurative meningitis localized to the basilar region. Microscopic examination of the organism from a culture of the cerebrospinal fluid withdrawn the previous day showed gram-negative diplobacilli which fermented maltose and dextrose but not lactose. The pus secured at necropsy revealed identical results, which were found on examination of the fluid from the lumbar puncture.

BACTERIOLOGY

A detailed discussion of the morphology and pathogenicity of the pneumobacillus is beyond the scope of this paper, but I shall try to describe it briefly and also to show how it is often confused with other organisms.

The pneumobacillus (Friedlander's) belongs to the *Bacillus mucosus* group [6] frequently present in pneumonia as a secondary invader, from 5 to 10 per cent, and is gram-negative, as are the other bacteria of the same group. Although rarely present, its effect is extremely severe and usually fatal. By the precipitin method of von Eisler and Porges [7] it must be differentiated from the bacilli found in ozena and rhinoscleroma which belong to the same group and which are also gram-negative and capsulated. The other two gram-negative encapsulated bacilli are *Bacterium lactis-aerogenous* of Escherich, who definitely recognized this organism found in the intestinal tract of young children, and *Bacterium acidi-lactici* of Huepe. The former ferments dextrose, saccharose and lactose; the latter ferments dextrose and lactose but not saccharose. The pneumobacillus ferments dextrose and saccharose but not lactose.

The pneumobacilli may be single, in diploform or in short chains. They are nonmotile, possess no flagella and form no spores. This bacillus is characteristically surrounded by a well developed capsule which is most perfectly demonstrated in preparations taken from some animal fluid, such as the secretion or exudate from infected areas. Figures 1 and 2 have been obtained from a blood culture and spinal fluid, the latter from a direct smear or culture. When seen in chains or groups, several bacilli may be enclosed in one capsule. Capsules may often be seen when more intense aniline dyes are employed, such as that of the Muir stain, Welsch's, Hiss's or Huntoon's.

The members of the *Bacillus capsulatus* group are thus easily differentiated from *Bacillus coli* or various paratyphoid bacilli which

6. Ford, W. W.: Text-Book of Bacteriology, Philadelphia, W. B. Saunders Company, 1927, p. 556.

7. Von Eisler, M., and Porges, O.: Ueber die Differenzierung der Kapselbakterien mit Hilfe agglutinierender und präzipitierender Immunsera, Centralbl. f. Bakteriol. **42**:660, 1906.

are methyl red positive, Voges-Proskauer negative, and permanently acid on sugar mediums.[8]

The pneumobacillus must also be differentiated from the *Pneumococcus mucosus* or type III pneumococcus[9] with which it is often confused. True, the latter has a richly developed capsule and is also attended by a high mortality but it differs from the pneumobacillus as follows: 1. It is a coccus. 2. It is gram-positive. 3. It is soluble in bile,[10] the active solvent agent in the bile being cholic acid. The workers in the Hospital of the Rockefeller Institute report "that among several hundred strains of pneumococcus isolated by us from lobar pneumonia none has failed to be dissolved by bile."[11] Pneumococci killed by heat are nonbile soluble. Neufeld and Handel,[12] however, have

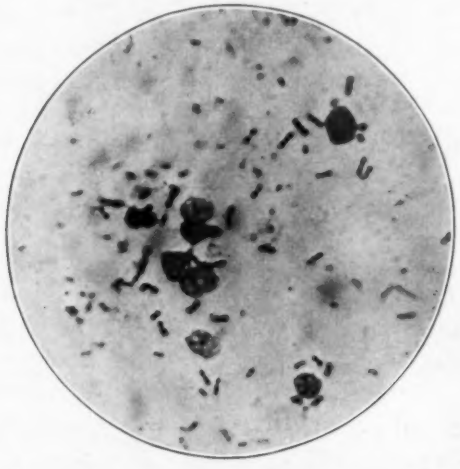

Fig. 1.—Photomicrograph of a smear from cerebrospinal fluid, showing mixed infection, streptococci with diploform pneumobacilli and leukocytes.

found that avirulent strains of pneumococcus are not dissolved by bile. 4. The pneumococcus grows characteristic colonies on blood.

Besides being a member of a heterogeneous group, there seems to exist different strains of the organism as reported by Julianelle,[13] who

8. Ford (footnote 6, p. 357).

9. Jordan, Edwin O.: General Bacteriology, ed. 8, Philadelphia, W. B. Saunders Company, 1926, pp. 225-227.

10. Neufeld: Ztschr. f. Hyg. **34**:454, 1900.

11. Avery, O. T.; Chickering, H. T.: Cole, R., and Dochez, A. R.: Monograph no. 7, Rockefeller Inst. Med. Res., Oct. 16, 1917, p. 14.

12. Ford (footnote 6, p. 387).

13. Julianelle, L. A.: Bacterial Variation in Cultures of Friedlander's Bacillus, J. Exper. Med. **47**:889 (June) 1928.

identified typical colonies of Friedlander's bacillus and classified them as the smooth (S) and the variant colonies rough (R), the organism in each instance differing not only morphologically, but biologically and antigenically. He ascribes this variation in culture to a certain dissociation phenomenon. The most salient differences are (*a*) that the S cells form colonies with a smooth surface; they are virulent, encapsulated and type specific; they produce the soluble specific substance. (*b*), The R cells, on the other hand, form colonies with a rough surface; they are avirulent, capsule-free and nontype specific, but species specific; they do not elaborate the soluble specific substance.

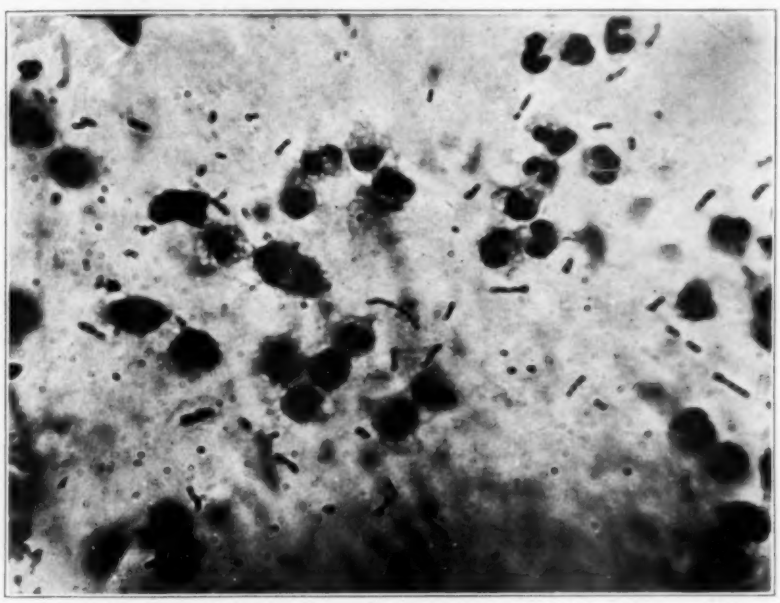

Fig. 2.—Photomicrograph of smear from cerebrospinal fluid, showing diploform pneumobacilli and some evolutionary forms.

According to Julianelle, the R forms of Friedlander's bacillus may be derived from the S strains by aging or by growth in the anti-S serum of the homologous type; the R strains may be isolated directly from the infection. It is an interesting observation that in the cases he studied, R variants were found only in chronic infections and always in conjunction with the S forms. Megrail [14] claimed that the younger the culture, the more toxic is its strain.

The cases of meningitis here reported as well as those reported by the French workers were secondary to otitis media. Although it is

14. Megrail, E.: Toxicity of Filtrates of B. Friedlander, Proc. Soc. Exper. Biol. & Med. **23**:655 (May) 1926.

claimed [15] that primary acute meningeal infection is due chiefly to two organisms, *Micrococcus intracellularis-meningitidis* and pneumococcus, Menetrier and Fontaine [16] reported a case of primary pneumobacillus meningitis:

A man, aged 33, previously in good health, with no history of ear disease or pulmonary infection, suddenly fell unconscious during his work. He was brought to the hospital in a state of coma, and died four days after admission. Repeated lumbar punctures failed to disclose the organism. Only necropsy revealed a diffuse suppurative leptomeningitis of the convexity and base. All other organs were searched for the portal of entrance, but none was found. Direct examination of the meningeal pus disclosed an encapsulated gram-negative diplococcus

Fig. 3.—Photomicrograph of long chained Streptococcus mucosus-capsulatus, from a blood culture.

bacillus in every way resembling Friedlander's pneumobacillus. Histologic pictures of the infected meninges and along the vessels of the meninges showed a suppurative reaction with the presence of pneumobacilli in abundance.

In the majority of cases thus far observed, the disease progressed so rapidly that in spite of early diagnosis and early surgical intervention death could not be averted.

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15. Hiss-Zinsser and Russell: A Text-Book of Bacteriology, ed. 5, New York, D. Appleton & Company, 1925.

16. Menetrier, P., and Fontaine, Bertrand: Pneumobacillus Meningitis, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:114 (Feb. 1) 1924.

REPORT OF CASES

CASE 1.—*History.*—H. R. P., aged 5 years, had a cold in his head for two days as a result of taking off his shoes and stockings in the yard. On October 18, at 2 p. m., he suddenly awakened from a sound sleep crying because of severe pain in his right ear. He looked flushed and feverish. He was then brought to the infirmary, where a paracentesis was done on his right drum under nitrous oxide anesthesia, as the drum was red and bulging, but hardly any pus was obtained. He had no tenderness over the mastoid. On Sunday morning, October 20, the patient began to complain of severe frontal headache, which was relieved after he had vomited. He continued to vomit all day. In the evening, he was restless and could not sleep. His temperature was 103 F. By Monday noon, the temperature rose to 105 F., and the patient "suddenly turned blue all over," but did not feel cold. He had no discharge from his ear. At this time, he complained that he could not see well and was somewhat stuporous. A physician was called to see the child, and because of the high temperature he performed a paracentesis on the left ear. No discharge was obtained. At 7 p. m., the patient's neck was stiff and extended. The family physician was called in to see the child; after calling another physician in consultation, a lumbar puncture was done. This revealed a great increase in cells. On Tuesday afternoon, October 22, the patient was brought to the infirmary in a stuporous state, though conscious.

Examination.—The patient was found in bed with the neck rigid and extended; the knees were flexed. The patient was rational. Examination of the chest gave negative results. The right ear looked well; there was no discharge, but the child was totally deaf, as shown by testing the left ear with a noise box. He showed spontaneous horizontal and rotatory nystagmus of marked amplitude to the left; a bilateral Kernig sign was present; abdominal and knee reflexes were absent. Examination of the eyegrounds showed the veins full, but no papilledema. The temperature was 102 F.; the pulse rate 96, and respirations, 48. The blood count showed: red cells, 4,500,000; hemoglobin, 96 per cent; white cells, 28,000; polymorphonuclears, 94 per cent, and lymphocytes, 6 per cent. The Wassermann reaction of the blood was negative. Lumbar puncture showed markedly increased pressure, turbid cerebrospinal fluid, no sugar and globulin, plus. Smear from the spinal fluid showed *Streptococcus mucosus-capsulatus.* Because of the high temperature, observations on lumbar puncture and total deafness in the right ear with spontaneous nystagmus, a diagnosis of septic leptomeningitis with concomitant acute diffuse labyrinthitis was made, and the patient was at once taken to the operating room.

Operation.—With the patient under a general anesthesia a simple and thorough mastoidectomy was done, because of the extensive necrosis of the mastoid intercellular tissue. Ossicles were removed, and also almost the posterior one third of the petrous bone. The solid angle of the labyrinth was then drilled through as a means of approach to the vestibule. A similar opening with the drill was made through the promontory. These openings were seen to communicate, but they contained no fluid. The cortex over the posterior fossa was then uncovered for about half an inch behind the lateral sinus. In that region, the dura was found somewhat red and tense. Incision was made through the dura, and considerable cerebrospinal fluid gushed out from the posterior fossa. After a rubber drain was placed from the mastoid antrum outside, the wound was filled with petrolatum and dressed.

Necropsy.—The brain filled the cranial cavity. On removal of the dura, the cerebral convolutions were completely flattened out. There was thick pus along the vessels over both hemispheres. At the junction of the anterior and inferior surfaces of the frontal lobes along the median border, there was thick exudate. The optic nerves were covered with fibrinous exudate which extended to the entire brain stem. There was no pus in the subdural space (it apparently had not one point from which it had spread). The pus appeared to follow the basal cisterni and entered the interior of the brain in the region of the third nerve on both sides. All markings of the brain stem were obliterated. The choroid plexuses looked gangrenous and had a bluish-black appearance. The third ventricle was distended and full of pus. The fourth ventricle was enormously distended and full of pus. The foramina of Luschka and Magendie were blocked. There appeared to be more pus in the ventricular system than on the cortex. The portal of entrance or path of infection could not be determined macroscopically.

Comment on Necropsy.—The patient had no papilledema and no retinal changes. The pupils were contracted and did not react to light yet the patient was blind for three days. This would indicate pus in the ventricle (Dr. Eagleton).

Comment.—The clinical picture, laboratory examination and necropsy show that this case was one of general leptomeningitis, of such severe nature that surgical intervention proved of no avail. As pointed out by Eagleton,[17] when the ventricles become affected, the process is terminal and is beyond operative intervention.

CASE 2.—*History.*—F. B., a girl, aged 4 years, was brought to the clinic on Nov. 6, 1929, giving a history of otorrhea from her right ear for four months, and since November 5, the pain in the right ear had been so severe that she could not sleep. Her temperature was 104.4 F.; the pulse rate, 140.

Examination.—There was otorrhea of the right ear, thick, yellow and foul-smelling pus being discharged, a perforation in the postero-inferior quadrant, postauricular swelling and some tenderness. The ear stood out from the head. The hearing was good. A diagnosis of acute exacerbation of chronic purulent otitis media was made, and the patient was admitted to the infirmary for operation. The child was undernourished, pale and smaller than one 4 years of age. Tonsils and adenoids were present, but were not large or infected; the lungs and heart were normal. Urinalysis gave negative results. The eyegrounds were normal. The blood count was: red cells, 3,000,000; hemoglobin, 45 per cent; white cells, 9,400; polymorphonuclears, 81 per cent; lymphocytes, 18 per cent, and transitionals, 1 per cent.

First Operation.—At 8 p. m. the same evening, the patient was taken to the operating room. A simple mastoidectomy was planned, but during the exenteration of the mastoid cells it was found that the necrosis of the intercellular tissue extended behind toward the lateral sinus, not only exposing the latter, but bathing the entire sigmoid portion of the lateral sinus in pus. The antrum was found high up and filled with greenish, cheesy pus. The wall of the sinus did not appear healthy, and so an incision was made into it, but no bleeding was obtained. The incision was then extended to the knee above and downward as far as the jugular bulb. The sinus was laid open, and a well organized infected clot of about 1

17. Eagleton, Wells P.: Physiological Factors in Control of Otitic Meningitis, Laryngoscope **37**:113 (Feb.) 1927.

inch was removed, after which bleeding was established from above and below. Three cotton-tailed packs were placed over the sinus wall. One suture was taken high up at the original incision and a rubber drain inserted. The internal jugular vein was not ligated.

Postoperative Diagnosis.—Acute mastoiditis with perisinous abscess and sinus thrombosis was diagnosed.

Course.—The following day the patient's temperature rose to 105 F., with typical intermissions. On November 8, the temperature rose from 105 to 106 F., and the patient did not look well. She was very restless and irrational, and picked at the bed clothes. The blood count was: erythrocytes, 3,100,000; hemoglobin, 55 per cent; white cells, 13,000; polymorphonuclears, 80 per cent; lymphocytes, 20 per cent. Blood culture revealed gram-negative bacilli pneumobacilli. It was decided to ligate the right internal jugular vein and give a transfusion. Examination that day revealed no headache, neck rigidity or Kernig sign. Examination of the eyegrounds showed the veins to be somewhat full, but not tortuous, and the disks clear. On November 9, the right internal jugular vein was ligated and resected above the facial nerve, and the upper end of the jugular vein was sewed up to the skin according to the Alexander method. The mastoid wound was reexamined and washed out with hydrogen dioxide; the packings over the sinus were not disturbed. A second transfusion of 100 cc. was given. While the patient was on the operating table, a lumbar puncture was done, and 8 cc. of fluid was withdrawn. The fluid was under marked pressure, but clear. There was reduction with Fehling's solution and no increase in cells; no micro-organisms were found from a direct smear of the cerebrospinal fluid. On November 10, the patient showed no signs of improvement. The temperature was 104 F. and still intermittent; the pulse rate, 120, and respirations, 24. The tongue was dry. There were herpes on the lips and sordes on the teeth. The neck was rigid, and there was a bilateral Kernig sign. Blood culture showed gram-negative bacilli—pneumobacilli. The patient was now transferred to the head division and, in view of the foregoing observations, exploration for meningitis was decided on. On November 11, the temperature varied from 98.6 at noon to 104 F. at 6 p. m.; the pulse rate was 140; respiration, 24. The blood count showed: erythrocytes, 3,500,000; hemoglobin, 70 per cent; white cells, 11,200; polymorphonuclears, 70 per cent, and lymphocytes, 30 per cent. The blood culture showed pneumobacilli. Because of the patient's intermittent temperature and lack of further meningeal signs, Dr. Eagleton contended that the case was one of blood stream infection. At 11:30 p. m., however, exploration for meningitis was begun under ether anesthesia.

Third Operation.—The mastoid cells were found well cleaned out and the antrum open; the sinus was found incised for about one half inch along the sigmoid portion. A Whiting incision was then made over the skin, extending to the external occipital protuberance, and the bleeding points were stopped. The cortex over the right temporosphenoidal lobe was removed enough to uncover the dura in the middle fossa for about 1 inch above the tegmen tympani and from the zygomatic angle to the lateral sinus. The petrosal angle was then removed as far as the semicircular canals. The lateral sinus was exposed as far back as the torculi; the cerebellum below was exposed as well as the jugular bulb; the facial ridge was lowered, but the ossicles were left intact. The whole sinus from the jugular bulb to about 1 inch behind the knee of the lateral sinus was found gangrenous and contained free thick pus and blood clots. Incisions into the lateral sinus were carried up as far as the torculi and downward as far as the jugular

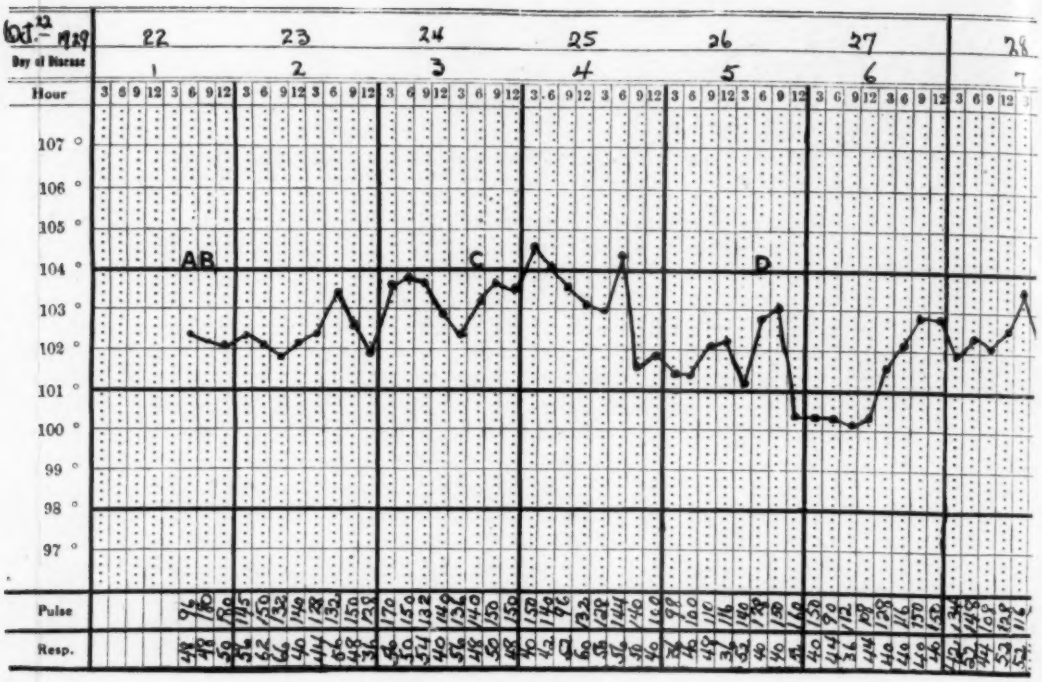

Figure 4—Left

Fig. 4.—Temperature chart in case 1. *A* indicates time of admis

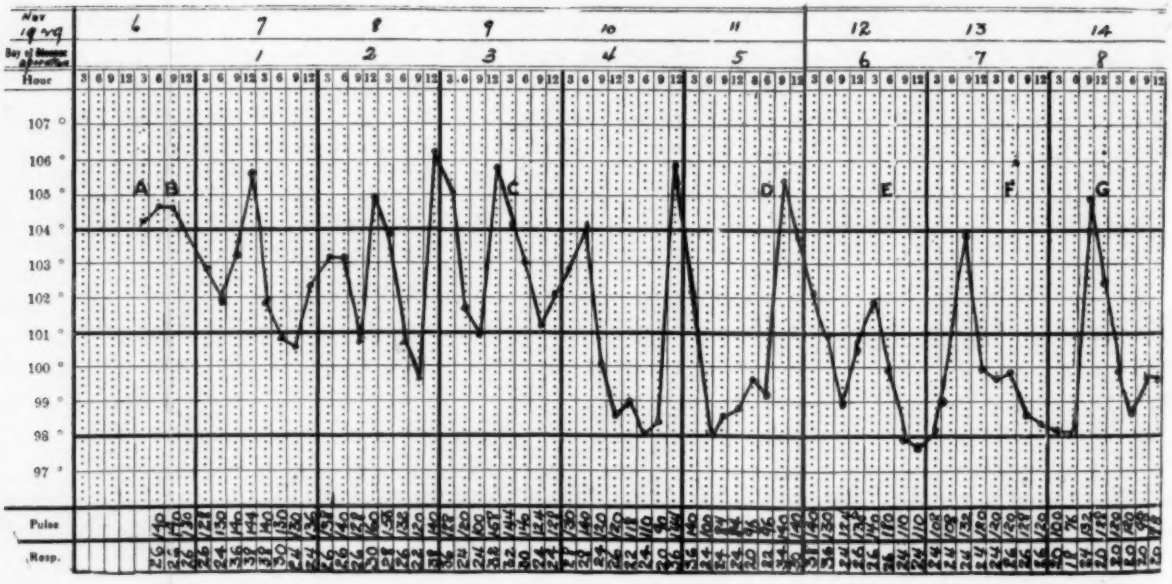

Figure 5—Left

Fig. 5.—Temperature chart in case 2, showing blood stream infection—septic sinus thrombophlebitis. *A* exploration for meningitis; *E*, *F*, *G* and *H*, transfusions.

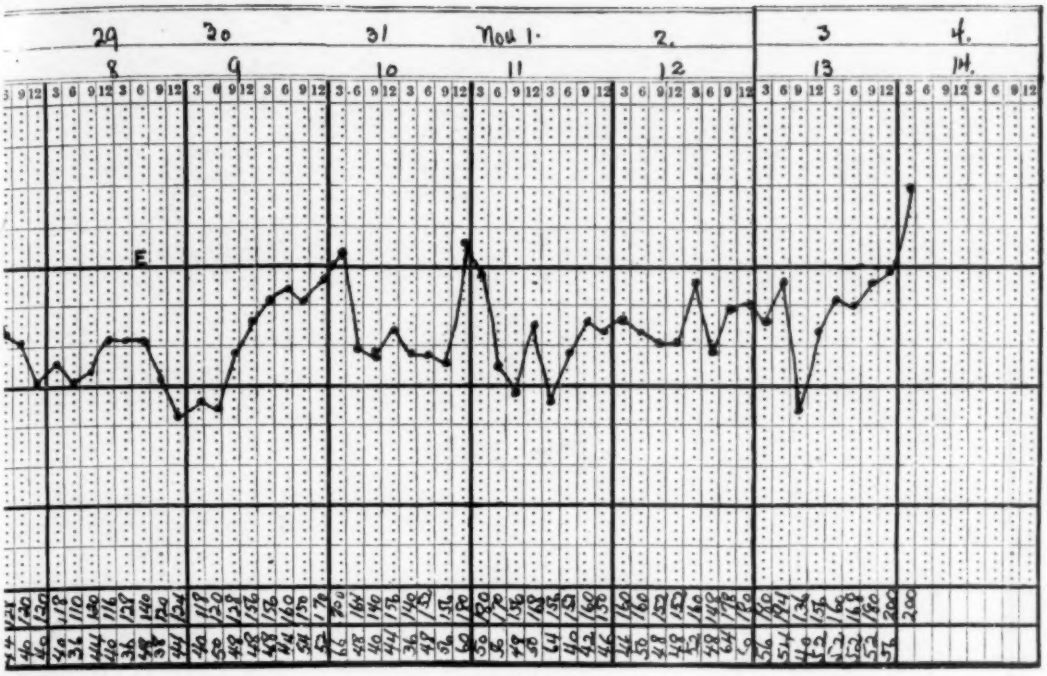

Figure 4—Right

sion; *B*, operation and transfusion; *C*, *D* and *E*, transfusions.

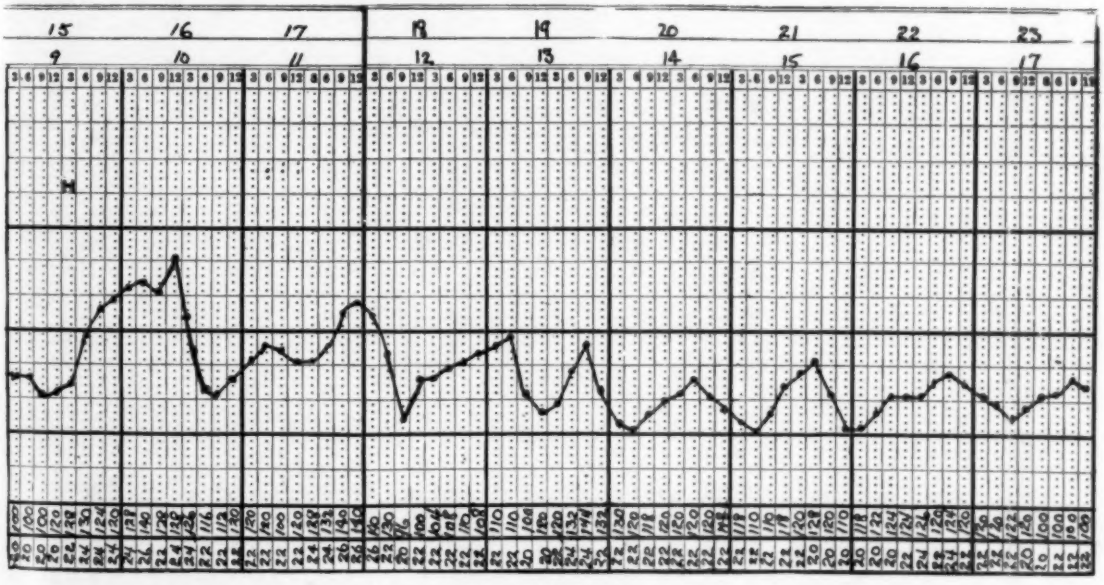

Figure 5—Right

indicates time of admission; *B*, time of operation; *C*, ligation of jugular vein; *D*, second operation,

Notes on Postoperative Progress

Date	Temperature	Pulse	Respiration	Blood Counts	Blood Cultures	Lumbar or Occipito-Atloid Punctures		Condition of Patient
October 23	102.0 103.4	140 150	56 66	White cells.....25,200 Polymorphonuclears..92% Lymphocytes.....4% Mononuclears.....4%	.....	L. P.	Fluid under pressure and turbid; 14,000 polymorphonuclears per c.mm.; no reduction with Fehling's solution; pneumobacilli	Poor; restless and picks at bed clothes; delirious at times and noisy, does not seem to see
October 24	103.8	150	50	.....	Negative, 18 hours	L. P. (a.m.)	Fluid under pressure and turbid; no reduction with Fehling's solution; 6,800 cells per c.mm.; coagulable albumin and globulin plus; pneumobacilli	Restless but rational
						O. A. (p.m.)	Fluid under pressure and turbid; 1,980 cells per c.mm.	
October 25	104.6	150	40	.....	Negative, 18 hours	O. A. (a.m.)	Fluid still under pressure; 980 cells per c.mm.; abundant pneumobacilli	Drowsy and cyanotic; has a general blood stream infection
October 26	101.4 103	98 150	36 50	.....	36 hours, gram-negative bacilli — pneumobacilli	L. P. (a.m.)	Fluid still turbid; coagulable albumin and globulin plus; no sugar; 16,000 cells per c.mm. abundant pneumobacilli	Very delirious and talkative; sight seems to clear; considerable twitching of face and entire body for one hour
October 27	100.4 102.8	150	40	.....	60 hours, gram-negative bacilli — pneumobacilli	L. P. (p.m.)	Not analyzed	Restless and entire body twitches; convulsive and loquacious; cannot see, claims "it's dark"
October 28	102.0 103.0	134 120	42	.....	.....	O. A. (a.m.)	Fluid turbid; no reduction with Fehling's solution; coagulable albumin and globulin plus; 430 cells per c.mm.; abundant pneumobacilli	Pupils contracted; cannot see
October 29	101.0 102.2	118 140	40	.....	24 hours, gram-negative bacilli — pneumobacilli	L. P.	No fluid obtained through needle	Still cannot see; pupils do not react to light; much worse; profoundly septic
October 30	100.4 103.8	118 170	.. 52	.....	24 hours, gram-negative bacilli — pneumobacilli	L. P.	Fluid too thick; not obtained	Twitches and is restless; pupils contracted and cannot see
October 31	104.2 101.6	200 156	64	.....	.....	.....		Perspires a great deal; condition poor
November 1	105.0 100.6	180 156	64	.....	.....	.....		Perspires a great deal; no change
November 2	102.2 103.6	160	48	.....	.....	L. P.	No fluid obtained.....	Respirations deep, shallow and irregular; cyanotic; twitching of face and body
November 3	102.6 104.0	180 200	56	.....	.....	L. P.	No fluid obtained.....	No change in condition
November 4	106.0	200	..	.....	.....	.....		Ceased breathing at 3 a. m.

bulb; the sinus was laid open, bleeding established and a rubber drain inserted into the jugular bulb; the hemorrhage was controlled. At this time a lumbar puncture was done; the fluid was found under pressure, but fairly clear, with a cell count of 180 per cubic millimeter. There was no reduction with Fehling's solution. This indicated subarachnoid drainage, and so incision of the dura was made in the posterior fossa just internal to the lateral sinus, whereupon there was a gush of cerebrospinal fluid; the dura in the middle fossa was similarly opened, and here also the cerebrospinal fluid gushed out. After two silk sutures were taken over the Whiting incision to hold the cotton packs in place, the wound was filled with petrolatum and covered with rubber smeared with petrolatum, and a starch bandage was put on. On November 12 and 13, the blood culture was still positive, with abundant pneumobacilli present. The temperature ranged from 102 to 104 F. The pulse rate was 140. Another transfusion was given, but the patient did not seem to show any improvement. On November 13 she appeared drowsy and fidgety, and the neck was somewhat rigid. On November 14, she had a chill with a rise in temperature to 105 F. at 9 a. m. Blood culture was positive for pneumobacilli. Another transfusion was given on November 15; the patient did not look well, though the temperature stayed down to 99 F. The blood culture, however, continued to be positive for pneumobacilli; the temperature rose again. Another lumbar puncture was done, and 15 cc. of fluid was withdrawn. The fluid was found clear and under no pressure; there was normal reduction with Fehling's solution, a trace of coagulable albumin, and no increase in globulin. The cell count was 2 per cubic millimeter. No micro-organisms were found. Culture from the spinal fluid failed to show a growth. On November 18, however, in spite of a positive blood culture for pneumobacilli, the patient took a turn for the better. The temperature gradually subsided. No further surgical measures were attempted. Uninterrupted recovery then ensued, and the patient was discharged from the infirmary on Jan. 4, 1930.

Comment.—Although the patient had signs of meningeal irritation with a spinal count of 180 cells per cubic millimeter, there evidently was no diffuse process, but a localized meningitis from the septic sinus thrombophlebitis. As shown in figure 5, the temperature never remained high as in case 1.

The patient's slow and uncertain recovery was no doubt due to her poor condition as a result of the chronic disease. Despite the positive blood culture, organisms were never found in the spinal fluid, and the pneumobacilli must have undergone evolutionary changes from a virulent to an avirulent form. The strain was not toxic enough to cause a generalized leptomeningitis.

SUMMARY

1. The pneumobacillus, although rare, is a virulent organism.

2. It possesses characteristics of its own, and is easily differentiated from both the pneumococci and colon-form groups, as well as from members of its own group.

3. It is more often found as the offending agent secondary to otitis and pulmonary infections than primarily.

4. Despite its virulence, the prognosis is not necessarily unfavorable.

CONCLUSIONS

Clinically and pathologically, septic leptomeningitis due to the pneumobacillus resembles in every way the fulminating type due to the pneumococcus; while cases due to the latter end fatally, those due to the pneumobacillus, if checked early, may be cured. It is therefore of importance to differentiate these two organisms.

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PHLEGMON OF THE NECK AND OSTEOMYELITIS OF THE MANDIBLE FOLLOWING TONSILLECTOMY*

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A phlegmon of the neck following tonsillectomy is itself not a common casualty. Kofler of Vienna in a recent statistical study found six phlegmons reported in six thousand tonsil operations. More have been reported in this country. I have been unable to find mention in the literature, however, of an osteomyelitis of the jaw in connection with a tonsillectomy.

REPORT OF A CASE

A girl, aged 18, suffered from repeated attacks of tonsillitis for which tonsillectomy was carried out under local anesthesia on June 4, 1929. The patient stated that the operative procedure was painless, little bleeding occurring during or after the operation, and that she felt nothing during the convalescence other than the usual discomfort while eating or swallowing. However, a week after the operation the jaw became locked in the region of the right tonsil. Hot applications were applied, but the pain increased, and the jaw became more rigid. As her condition became more critical, she was taken back to the hospital. This occurred a week after the onset of the unfavorable symptoms and two weeks after the tonsillectomy. At this time the attending physician, using gas anesthesia, tried to open the jaw in order to lance from the inside what appeared to be an abscess. Following this procedure, her temperature rose to 105 F., and she had chills. The rigidity of the jaw did not improve, and swelling of the right submaxillary region was more marked than before. Two days later the patient came under my care, and the following observations were made:

The temperature was 103 F., and the pulse rate 120. The patient was in a somnolent condition and hardly responded to questions. The right side of the face was shiny, swollen and red; edema extended from the right side of the forehead to the sternoclavicular portion. The right eye was completely closed. Infiltration was most marked in the submaxillary region and in front of the sternocleidomastoid muscle, where fluctuation was detected. Examination of the throat was impossible on account of the trismus, which was so pronounced that the broad side of a wooden tongue depressor could not be wedged between the upper and lower teeth. The odor from the mouth was foul.

An incision was made at the point of the most marked infiltration, about 2 fingerbreadths below the margin of the mandible in front of the sternocleidomastoid muscle. With blunt forceps the fascia and the muscular structures were penetrated. About 1.5 cm. deep a pocket of pus was opened, and 2 ounces (59 cc.) of yellowish pus with a colonic odor escaped. On palpation of the cavity the lower part of the mandible, bare of its periosteum, could be felt. This cavity reached up to the hypopharynx, so that there was direct communication between the pocket of pus and the mouth. It was also possible to palpate the right pterygoid process of the sphenoidal bone. Two soft rubber drains were inserted.

* Submitted for publication, March 8, 1930.

Four days after the operation, an x-ray picture of the lower jaw was taken because of the possibility of osteomyelitis, periostitis or purulent arthritis of the jaw. However, no disease of the bone was reported save for some bone rarification at the portion of the right mandibular angle. The patient improved gradually, and the temperature was normal five days after the operation. Profuse purulent discharge from the wound and the mouth continued, and there was little improvement as to the lockjaw, so that three weeks after the operation the patient was able to open the jaw only the width of 1 fingerbreath. A second roentgenogram was made at that time, and the following report was given:

"X-ray examination of the right lower jaw shows a marked abnormality of the portion between the horizontal and ascending rami, extending considerably up the latter. The appearance is that of a fracture line extending from about 1

Fig. 1.—The line of separation of the bone through the right ascending and the horizontal ramus of the mandible.

inch below the articulation of the posterior edge of the vertical ramus to about 1 inch anterior to the angle of the jaw of the inferior edge of the horizontal ramus. The bone anterior to this apparent fracture line, involving the adjacent portion of both rami shows apparently marked irregular absorption. This appearance is rather suggestive of an osteomyelitis with pathologic fracture due to weakening" (fig. 1).

These observations explained the persistent lockjaw, and the question was what line of treatment should be instituted in regard to the fracture. On palpation, that part of the jaw seemed to be solid, and no signs of complete through and through separation could be detected. The remaining intact bony structure was apparently strong enough to withstand the act of chewing. A course of observation was decided on, with a routine treatment for discharging wounds, irrigation with surgical solution of chlorinated soda, etc.

About two months after the operation, the patient was able to open the jaw to its full extent and to chew normally. The wound was still discharging. A roentgenogram taken on November 6 showed definitely a sequestrum at the angle of the jaw, and the bare bone could be felt. The sequestrum was still firmly attached to the healthy portion of the jaw, but further delay in removing the sequestrum did not seem justified, since the suppuration would not cease as long as the dead piece of bone remained, and there was no longer danger of injuring the firmness of the two united fragments by removing the splinter.

On November 18, under general anesthesia, an incision was made over the angle of the jaw through the sinus, and a large sequestrum at the angle of the jaw was removed (fig. 2). Ten days later, the wound ceased to discharge.

Fig. 2.—The sequestrum which was removed from the mandible. Its angle portion was mostly involved.

SUMMARY

From this history it is apparent that an infection of the wound bed began eight days after the tonsillectomy, which was too late for it to be ascribed to the surgical manipulation itself. I assume that one of the many saprophytic germs of the mouth became virulent and caused the infection. The rapidly spreading process involved the mandibular joint and produced rigidity of the jaw. Eight days after onset of the complication, in his effort to relieve the condition by incising the abscess from inside, the physician was forced to pry open the jaw in order to reach the tonsillar bed. At that time the structure of the bone was already affected by the inflammatory process, and this infection apparently involved the periostium in the region of the angle of the right mandible, with a subsequent osteomyelitis, followed by a separation of bone producing the sequestrum, which was removed 160 days after the tonsillectomy.

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TRANSPOSITION OF SECTION FROM THE AURICLE FOR THE CORRECTION OF A NASAL DEFECT

REPORT OF A CASE *

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The preservation of the vitality of tissue when transferred en masse from one part of the body to another without the aid of a connecting pedicle is always attended by uncertainty, even under the most favorable circumstances. If one is able to accomplish this, however, especially in the restoration of a part important not only from a functional but from an esthetic point of view, such as the nose, there is a great gain over the pedicled flap method, which leaves a formidable scar on the face that it is difficult to conceal. Besides, even the latter method is attended by a certain amount of risk as it is uncertain whether or not the transplanted tissue will remain alive after the pedicle has been severed, and there is always great inconvenience and discomfort to the patient, while the flap is establishing a vascular connection in its new position. However, the pedicled flap method for restoring defects in the nose or any other part of the body cannot be displaced altogether by that of transference of tissue en masse. Each case must be decided on its own merits, the chances of success being based largely on the size and location of the defect, the age of the patient, his general condition and the histologic structure and vascularity of the transplanted tissue as well as of the tissue adjacent to the part to be restored.

The marked uncertainty attending the transposition of soft tissues in contradistinction to that of bone and cartilage lies in the facts that the latter are slower in their metabolic processes, are more tolerant to traumatism and are capable of receiving their nourishment temporarily from the serous pabulum which surrounds them in their new position until new blood vessels, penetrating from the adjacent arterioles, insure their permanent nutrition. In the case of cartilage, which is nourished by intercellular circulation, the shock of transference is even less than it is in bone. Not so with soft tissues; these will submit to little interference with their blood supply, and when this is completely cut off, cellular death quickly occurs unless the circulation is restored almost immediately.

* Submitted for publication, April 10, 1930.

The foregoing paragraphs contain a brief summary of the practical clinical problem presented when it is desired to transfer soft tissue en masse from one part of the body to another with the expectation of preserving its viability.

The transposition of living soft tissue with preservation of its cellular activity and power of growth in its new position is a tribute of the highest quality to the technical skill, manual dexterity and good judgment of the surgeon.

Many conditions determine the success or failure of this operation. They involve chiefly the patient and his environment, the part of the body to be restored, the location from which the transplant is to be removed, the technic employed, the surgeon and his assistants and, fully as important as any other consideration, the after-treatment.

I shall consider here only the restoration of the nose after a part of its soft or semisoft tissue had been destroyed, and I shall report one of my cases in which this was successfully accomplished.

REPORT OF CASE

History.—A woman, aged 25, married, suffered an injury which resulted in the loss of a part of her nose. The history prior to this injury had no bearing on her present condition.

For two years she had noticed a small papule or pimple on the lower margin of her right ala nasi, which her physician said was a hematoma. She then consulted a radiologist, who attempted to remove the papule by means of electric needles. Shortly after the first and only treatment, that side of her nose became swollen and painful. This condition was followed by ulceration and sloughing of the entire thickness of the ala over an area extending from the tip down to its attachment to the lip; the lost portion was about one-third inch in width, leaving the nasal cavity exposed to view.

This accident happened about a year before the patient came to me. In the meanwhile, she had suffered great anguish on account of the deformity, and both she and her husband insisted that something should be done for her relief.

Examination.—Examination revealed a comely young woman, apparently in the best of health. There was a deficiency in the margin of her right ala nasi extending from the tip of the nose down to its attachment to the upper lip, exposing to view the mucous membrane of the nasal cavity. The margin of this defect was curved, the concavity pointing downward. It was deeply indurated and evidently contained much scar tissue. The septum was not injured, and, aside from this defect in the ala, the nose appeared to be normal.

I explained to the patient that the classic method for repairing such a deformity was by means of a pedicled flap thrown from the forehead, the cheek or the lip, but that more or less scarring would be inevitable. Both she and her husband objected to this, if it could possibly be avoided.

After thinking the matter over, I decided that a transplant of whole tissue from the auricle would be ideal if it could only be transposed without destroying its vitality, which I seriously doubted. However, I discussed the matter frankly with my patient, and, though I told her that the chances were about evenly divided between success and failure, she accepted the conditions enthusiastically.

My chief reasons for selecting the auricle were the following:

1. The youth and vigorous health of the patient would favor transposition of soft tissue en masse.

2. The auricle consists of two layers of skin and subcutaneous tissue, between which is a very thin layer of cartilage, and its free edge is not only covered with skin, but resembles closely the free edge of the ala nasi; a portion, therefore, could be selected that would almost duplicate in every way the missing ala.

3. This portion of the ear is not only quite vascular, but it is exposed to the air and has the same tint as the skin of the nose.

4. The proximity of the ear to the nose and the facility with which all of the preparations for the rapid transfer of the tissue can be made are important.

Fig. 1.—Patient before operation.

5. The removal of the section would not be followed by deformity of the ear. There would, of course, be a slight diminution in the size of the auricle, but it would not be noticeable unless special attention should be drawn to it.

Operation.—Previous Preparation: A plaster of paris mold was made to fit into the defect in such a manner as to represent the exact amount of tissue needed to correct the deformity. A pattern of this mold was then made in adhesive plaster. The pattern was made about one-third larger than needed, as transposed tissue always contracts. The pattern was then made to adhere to the portion of the right auricle selected for transfer. Great care was taken to choose a portion of the auricle that was in shape and thickness analogous to the corresponding unaffected ala.

In doing this work, it must be remembered that there are at least six essentials that are general in their application:

1. Strict asepsis must be observed.

2. The highest speed consistent with accuracy must be employed in the transference of the transplant to its new position.

3. All cutting instruments must have razor edges.

4. The transposed tissue must not be handled or contused in any way, and as few sutures as possible should be used.

5. The transplant must be kept continuously warm by means of the Carter hot water jacket after it has been placed in position.

6. The transplant must be fully one-third larger than is needed to cover the defect.

Technic: Tracheal gas-oxygen-ether anesthesia was employed. After the operative area had been swabbed with iodine, followed by alcohol, only physiologic

Fig. 2.—Patient six months after operation.

solution of sodium chloride was used. The margins of the nasal defect were pared to a depth that included the dense cicatricial tissue. This area was then covered with gauze soaked in warm physiologic solution of sodium chloride.

The tissue to be transplanted was then removed in the following manner: An incision was made along the edge of the adhesive plaster pattern which had been made to adhere to the posterior surface of the section of the auricle to be removed. This incision extended down to the cartilage.

Four curved eye needles, previously threaded with 000 Bauer and Black cutaneous sutures, were passed at appropriate intervals through the edges of the area incised, which was to be the anterior surface when the transplant was in position. The needles were left threaded, ready to be passed through the edges of the denuded defect in the nose.

The original incision was now carried through the cartilage, and, after a thin strip of this had been removed from the transplant, was extended through the remaining thickness of the auricle. The transplant, after being completely severed from the auricle, was quickly transferred to the defect in the nose and secured in

position by means of the four sutures already mentioned. No attempt was made to place sutures inside the nose as the inner edge of the transplanted tissue could be adjusted without additional traumatism by placing a light packing of petrolatum gauze in the nostril.

I wish to emphasize the fact that only four sutures were used; I regard this as important, for sutures interfere with blood supply; the fewer used, the better.

Attention was now directed to the defect in the auricle caused by the removal of the transplant. In the meantime, the nose was kept warm by means of gauze soaked in hot saline solution.

In closing the defect, I planned to reduce the size of the entire auricle uniformly and to make it as much like the other side as possible, only smaller. This I accomplished by removing an additional triangular piece, the apex of which reached

Fig. 3.—Dr. Carter's nasal water jacket.

almost to the external auditory meatus. I then brought the parts together accurately with skin sutures.

The patient was then removed to her bed and the nasal hot water jacket applied. This was kept on at a temperature of about 99 F. for forty-eight hours. (I devised and personally constructed this water jacket for the nose about eighteen years ago and presented it before the New York Academy of Medicine and other societies. I make this statement in support of my original idea, as this device has recently been attributed to another.)

The water jacket consists of a hollow copper splint shaped to fit over the nose. At either end of its lower extremity there is a small tube communicating with its interior; to these, rubber tubing may be attached. The inlet tube is connected with a fountain syringe which is kept filled with water kept at a temperature of 100 F. The bag is hung about a foot above the patient's head. The outlet tube, attached to the opposite side of the jacket, conveys the water into the waste pail after it has passed through the apparatus.

After the water jacket had remained in place for forty-eight hours, it was removed. Much to my surprise the transplanted tissue, though quite blue at first, remained alive. The skin assumed a normal color in about three weeks.

The deformity was somewhat overcorrected, for I wanted no tension on the sutures, and I knew that contraction would occur.

Healing was prompt and the difference in the size of the two ears would scarcely be noticed without close inspection.

Figure 2 was taken about six months after the operation. I saw the patient recently, and I found the condition of the nose entirely satisfactory.

CONCLUSION

I wish to emphasize the fact that the transference of soft tissue en masse should not be attempted unless a careful analysis of the case has previously been made, and unless on the basis of this analysis the conclusion has been reached that the exacting conditions I have outlined may be met with reasonable assurance.

2 East Fifty-Fourth Street.

PRIMARY BLASTOMYCOSIS OF THE TONGUE*

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Primary blastomycosis, without evidence of cutaneous or systemic involvement, is rare. Blastomycosis of the tongue is unusual; it may occur as a primary condition or as part of a general systemic infection. Reported herein is a case of primary blastomycosis of the tongue.

Only four cases of blastomycosis of the tongue have been reported in the literature. Ravogli,[1] Hoffmann,[2] and Copelli[3] each reported a case, in all of which, besides the lesion of the tongue, there was blastomycosis elsewhere in the body. The fourth case, reported by New[4] in 1917, was apparently a primary lesion, but the patient died thirty-one months after the onset of symptoms.

Five cases of primary blastomycosis of the larynx have been reported, two of which were reported by New in 1928.[5] A few other isolated cases of the lesion have been noted. Brewer and Wood[6] described blastomycosis of the spine without other manifestation of the disease; Campbell[7] described blastomycosis of the tibia, and Haase, Hall and Marshall[8] described blastomycosis of the epididymis. Lesions of the skin were present in the last two cases.

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* Submitted for publication, Jan. 31, 1930.

* From the Section on Laryngology, Oral and Plastic Surgery, the Mayo Clinic.

1. Ravogli, A.: Some Further Observations on Blastomycosis, Lancet-Clinic **15**:489 (May) 1911.

2. Hoffmann, E.: Chronische Blastomykose (?) der Haut und Zunge bei einem 40 jährigen Manne, Sitzungsb. d. naturh. Ver. d. preuss. Rhenil. u. Westphal., Bonn. 1911-1912, B, p. 20.

3. Copelli, M.: A Case of Blastomycosis, J. Cutan. Dis. **31**:51 (Jan.) 1913.

4. New, G. B.: Blastomycosis of the Tongue, J. A. M. A. **68**:186 (Jan. 20) 1917.

5. New, G. B.: Blastomycosis of the Larynx, Ann. Otol. Rhin. & Laryng. **37**:240 (March) 1928.

6. Brewer, G. E., and Wood, F. C.: Blastomycosis of the Spine, Ann. Surg. **48**:889 (Dec.) 1908.

7. Campbell, J. L.: Systematic Blastomycosis, Atlanta Jour.-Rec. Med. **12**: 308, 1909.

8. Haase, Marcus; Hall, E. R., and Marshall, C. H.: Local Blastomycosis, J. A. M. A. **79**:820 (Sept. 2) 1922.

Numerous cases of cutaneous blastomycosis, the commonest form of the disease, have been observed since Gilchrist,[9] in 1894, reported the first case.

Systemic blastomycosis was first described by Walker [10] and Montgomery. It occurs most often in males; thirty-four of Stober's [11] series of thirty-six cases were in men. The disease was usually seen in the third and fourth decades of life, and poverty, unhygienic surroundings, exposure and hard manual labor seemed to be contributing factors. Trauma, even if only a slight abrasion, may predispose to infection, especially of the skin. It was Stober's belief that it may be spread by

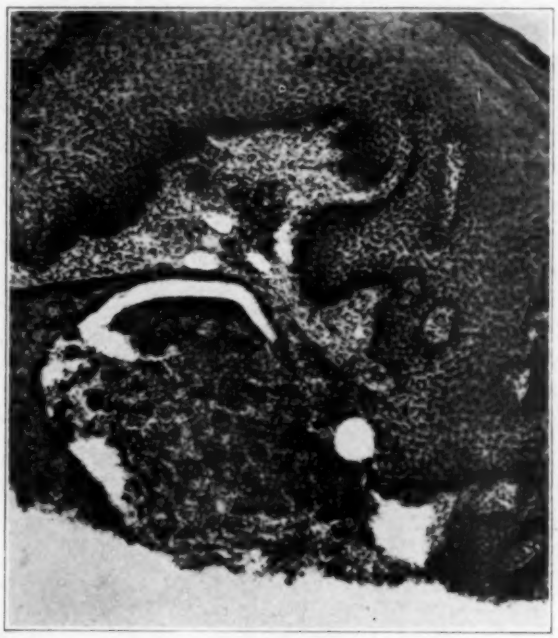

Fig. 1.—Minute epidermal abscess.

dust or droplet infection, the respiratory tract being the usual atrium in systemic cases.

The causative organism is a yeast fungus, fairly readily found in a darkened microscopic field, in minute epidermal or visceral abscesses

9. Gilchrist, Casper: Blastomycetic Dermatitis in the Negro, Brit. M. J. **2**:1321, 1902.

10. Walker, J. W., and Montgomery, F. H.: Further Report of a Previously Recorded Case of Blastomycosis of the Skin, Systemic Infection with Blastomyces; Death, Autopsy, J. A. M. A. **38**:867 (April 5) 1902.

11. Stober, A. M.: Systemic Blastomycosis, Arch. Int. Med. **13**:509 (April) 1914.

or in sputum of patients with pulmonary disease. The organisms are double-contoured, homogeneous, round or oval bodies with an outer refractile capsule, which appear in pairs or clumps. In the tissues, they multiply by budding, from which they are termed blastomyces.

In the skin the characteristic manifestations are marked epithelial hyperplasia with minute epidermal abscesses and a peculiar granulomatous infiltration of the corium. Typical cutaneous lesions often develop at the site of these abscesses. The skin of the face, forearms, hands and legs is more often involved, and the cutaneous lesions are somewhat irregular, with a papillomatous or verrucous surface, and a raised indurated border containing the miliary abscesses. The lesions slowly extend centrifugally and leave a thin scar in the healed areas.

Stober, and Haase, Hall and Marshall showed that the organisms were spread by the blood stream, hence any organ or tissue may be affected. The extent of dissemination probably depends on the time the infection exists in the body. Howes and Morse [12] stated that involvement of the lung is found in 97 per cent of cases that come to necropsy. Besides the lung, there may be lesions in the mucous membrane of the larynx or trachea, and abscesses may be found in the viscera, bones, muscles, lymph nodes and subcutaneous tissues.

Blastomycosis should be considered in all cases of chronic ulcerative lesions of the skin, deep abscesses, suppurative disease of the bone, and in protracted diseases of the lung which simulate tuberculosis. The systemic infection must also be distinguished from staphylococcic or streptococcic septicemia.

Epithelioma is likely to be confused with blastomycosis, and microscopic examination should be the deciding factor, even though the diagnosis of epithelioma may be assumed from a lesion which tends to grow more slowly and exhibits greater induration of the adjacent tissues.

To rule out tuberculosis, the examination of sputum in pulmonary disease and of the pus from the abscesses in cases of involvement of bone or viscera should decide the issue.

Roentgenograms in cases of osteoporosis, of periosteal thickening in syphilitic lesions of the bones and of blastomycosis may be identical, and gumma is likely to be confused with blastomycosis of the skin or viscera. The Wassermann reaction of the blood, clinical data and the microscopic examination should establish the diagnosis. One should not be misled by the improvement which follows the administration of potassium iodide.

Coccidioidal granuloma is distinguished by the greater tendency toward lymphatic involvement, the skin lesions which are more severely

12. Howes, W. B., and Morse, P. F.: Report of Two Cases of Blastomycosis, Boston M. & S. J. **185**:315 (Sept. 15) 1921.

ulcerated, shorter duration of the disease and the fact that reproduction in the tissues occurs by the formation of endospores.

The prognosis in blastomycosis of the skin is not particularly serious, but in a systemic infection it is very grave in 90 per cent [13] of the cases. This may be due to the fact that the condition is not recognized until dissemination has become extensive. Howes and Morse stated that nine of every ten patients with systemic blastomycosis die within a year. Stober found that the duration of the illness varied from four months to two and a half years, death often resulting from secondary pyogenic infection.

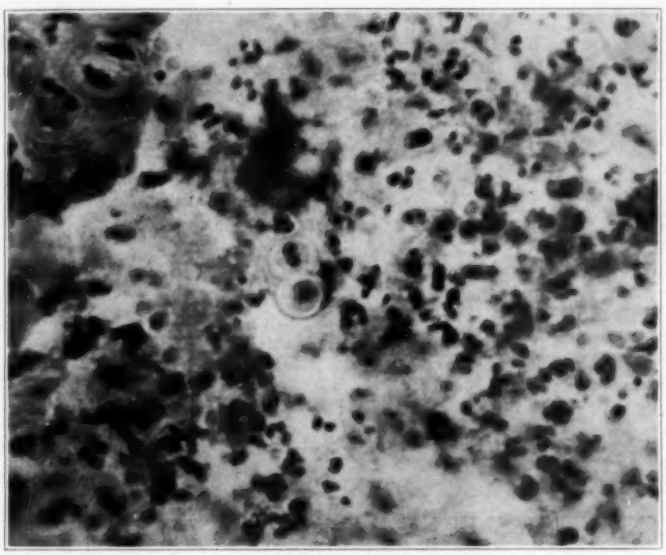

Fig. 2.—Blastomyces in the center of the abscess.

Attention should be directed to the environment, for unhygienic surroundings may have a significant bearing on the growth of the mold.[11]

The subcutaneous injection of filtrate of old cultures of blastomyces as a therapeutic measure was suggested by Stober. Roentgen ray, radium, surgical diathermy and large doses of potassium iodide are included in the treatment of the disease. New gives potassium iodide in doses of 10 drops, increased 3 drops a day until 200 drops are given three times a day.

The case reported here is the second case of primary blastomycosis of the tongue to be reported from the Mayo Clinic. It differs from the cases of New and Copelli in that the lesion was far removed from

13. Langdon, J. F.: Blastomycosis, Nebraska M. J. **7**:172 (May) 1922. Strober (footnote 11).

the area of lingual tonsil, being situated on the ventral surface of the tongue anteriorly.

REPORT OF A CASE

A man, aged 54, was admitted to the clinic on July 5, 1929, complaining of a growth on the tongue. Three months before admission he had noted a sore spot on the left ventral surface of the tongue, which had gradually increased in size. It was moderately painful, but did not ulcerate and there was no bleeding. He had had difficulty in eating, and this had resulted in loss of weight of 5 or 10 pounds (2.3 or 4.5 Kg.). He had had several teeth removed, with some relief from the pain; he had made local applications and had received a few electrical treatments, without benefit.

The patient was a native of Ireland, but he had been living on a farm in North Dakota since he had come to this country three years before presentation. He

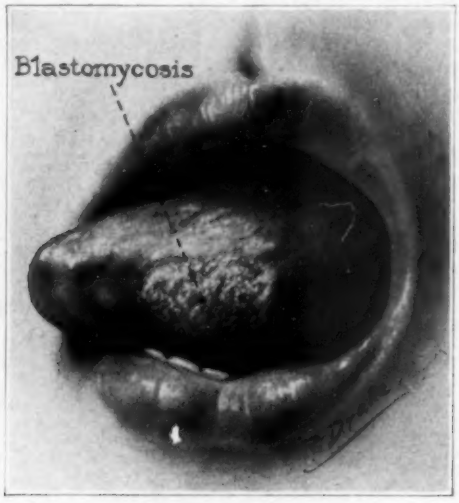

Fig. 3.—Blastomycosis of the tongue.

had chewed and smoked tobacco moderately prior to the onset of the present trouble.

Examination disclosed a superficial, moderately firm, nonulcerated lesion which looked like a low grade, papillary epithelioma, on the left ventral surface of the tongue. It measured 2 by 2.5 cm. A few septic snags of teeth were found; the tonsils were atrophic, and the throat was injected. A few slightly enlarged left submaxillary lymph nodes were palpable. A slight deafness of the left ear was elicited. The left eye, which had been weak and sore before the regional teeth were removed, was normal. The patient was moderately emaciated. Peripheral sclerosis was graded 1, and sclerosis of the hypertension type of the retinal arteries was graded 1. The right inguinal ring was relaxed. Roentgenograms of the thorax, an analysis of the blood and urine and the Wassermann reaction of the blood were negative.

A biopsy of the lesion of the tongue immediately preceding operation showed blastomycosis. The lesion was destroyed with diathermy. The wound was filled in with granulation tissue and was healed in a few weeks.

Primary involvement of the tongue by the blastomyces probably is a more common occurrence than is generally supposed. It is conceivable that such a case may be mistaken for a syphilitic lesion, which responds well to the iodides, or it may be mistaken for epithelioma, on account of the gross appearance and the microscopic signs of marked epithelial hyperplasia and granulomatous infiltration.

STATISTICAL STUDIES OF THE CHILDREN IN PUBLIC SCHOOLS FOR THE DEAF

ADDITIONAL REPORT OF COMMITTEE, DIVISION OF MEDICAL SCIENCES, NATIONAL RESEARCH COUNCIL *

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Our previous report [1] included results of the examination of 3,120 children in which the effort was made to collect statistics that would throw a light on some of the outstanding clinical problems relating to deaf children. The value of conclusions to be drawn from such statistical studies depends in no small measure on the number of children examined. Errors which are bound to creep in are in this way largely offset by the number of examinations. We were requested, therefore, by the National Research Council to pursue these studies in other public institutions for the deaf in order to bring the total number of children examined up to approximately 5,000. The previous studies included institutions located in the Middle West, that is, in Illinois, Missouri, Iowa, Indiana, Ohio, western Pennsylvania, Michigan, Minnesota and Wisconsin. Additional schools included institutions located in states on the eastern seaboard.

New York City (3 schools)	874 children
Eastern Pennsylvania (Philadelphia)	522 children
New Jersey (Trenton)	303 children
Virginia (Staunton)	199 children
North Carolina (Morganton)	330 children
Total	2,228 children

* Submitted for publication, April 12, 1930.

* The committee consisted of Dr. Charles W. Richardson, chairman (deceased), Dr. Wendell C. Phillips, Dr. George E. Shambaugh and ex-officio chairman of the Division of Medical Sciences.

1. Shambaugh, G. E.; Hagens, E. W.; Holderman, J. W., and Watkins, R. W.: Physical Causes of Deafness: II. Statistical Studies of the Children in the Public Schools for the Deaf, Arch. Otolaryng. **7**:424 (May) 1928; Reprint and Circular Series of the National Research Council, no. 82.

The addition of 2,228 children in these later studies brought the total up to 5,348 children examined. In this report we have analyzed these statistics by making use of a series of statistical tables.

The ultimate aim in these studies is to provide material which it is hoped may assist in efforts to ameliorate the sad plight of these children by throwing light on some of the clinical problems. We have made no effort to point out lines along which such efforts might profitably be directed. Such labors are left for others. We hope that our studies will assist in such work.

In our previous study of 3,120 children we found that 1,928 were congenitally deaf and 1,192 had acquired deafness. In our present

TABLE 1.—*Relation of Sex to Acquired and Congenital Deafness*

Congenital				Acquired			
Female		Male		Female		Male	
Number of Cases	Per Cent	Number of Cases	Per Cent	Number of Cases	Per Cent	Number of Cases	Per Cent
1,544	46.31	1,790	53.69	853	42.35	1,161	57.65

TABLE 2.—*Age at Which Deafness Was Acquired*

Age in Years	Number of Cases	Per Cent
Before 3	1,243	61.71
From 3 to 5	346	17.17
From 5 to 10	210	10.42
Over 10	28	1.39
Unknown	187	9.31

study of 2,228 children, 1,406 were congenitally deaf and 822 had acquired deafness; that is, of the 5,348 children 3,334 were congenitally deaf and 2,014 had acquired deafness. Of the total number of children with congenital deafness 1,544, or 46.31 per cent, were female and 1,790, or 53.69 per cent, male. Of the 2,014 with acquired deafness 853, or 42.53 per cent, were female and 1,161, or 57.65 per cent, male. It is seen, therefore, that sex does not play an important rôle. In both congenital and acquired deafness males predominate slightly over females.

In our previous studies we found that 706 of the total of 3,120 children acquired deafness before the age of 3. In our present studies there were 537 children who acquired deafness before this age. A total, therefore, of 1,243 children, or 61.71 per cent, acquired deafness before

3 years of age. These figures look rather large, and we cannot escape the conclusion that there is probably no small number of cases of so-called acquired deafness before the age of 3, which, if we knew the facts more accurately, should be classed as congenital. The reason for this conclusion is obvious to anyone who has had any experience in undertaking to gather accurate data from the parents who are always anxious to escape the opprobrium which they feel falls on them if they give birth to a deafened child. In many children who are deaf from birth the fact that they are deaf often becomes established only when they reach this age, and the parents are usually prone to believe that the child heard until they themselves discovered the fact that it was deaf. Our previous report included 193 children between the ages of 3 and 5, and our present studies 153, making a total of 346, or 17.17 per cent. Our previous studies included 144 children between the ages of 5 and 10, and our present statistics 66, making a total of 210, or 10.42 per cent. Of the children who had acquired deafness after the age of 10 our previous report included but 14. Our present studies also include 14, making a total of but 28, or 1.39 per cent. There were a number of children for whom we could get no definite statement as to the age when the deafness was acquired. Our former report included 135 such cases, and our present report 52. There is a total, therefore, of 187 children, or 9.31 per cent, in whom the age at which the deafness was acquired could not be ascertained.

A glance at these figures brings to light a fact that was commented on in our previous report, namely, that early childhood, or up to the age of 10, is the danger period for loss of hearing in children. In this connection it might not be amiss to call attention to the fact that after the age of 10 there is a period of approximately a decade or more when relatively few people acquire severe deafness. A statistical study of deafness acquired by adults would be of considerable interest. From observations made over many years of practice in otology, I am of the conviction that the many cases in which severe deafness was acquired in adult life, including the cases which we term otosclerosis, are those which rarely begin to develop before the age of 20 and which, as far as I have been able to determine, bear no relation to the ear troubles so prevalent in childhood, namely, those of the tubotympanic processes. Efforts are being made at present to establish regulations for the examination of the hearing in school children. Such examinations are highly to be desired for several reasons, but I have the impression that in some of the propaganda put forward to further these examinations a false

note has been struck; for it has been urged that by detecting slight defects in the hearing of children these examinations may lead to treatment in the incipient stages in persons in whom progressive deafness develops when they attain adult life.

Table 3 needs no particular comment, except that these statistics must not be taken to show that deafness is more prevalent in children of one nationality than in those of another, because the figures do not attempt to show the total number of children of each nationality.

ETIOLOGY OF ACQUIRED DEAFNESS

It is hardly necessary to point out that statistics on the etiology of acquired deafness represent one of the most important studies made in

TABLE 3.—*Relation of Nationality to Number of Cases of Deafness*

Nationality	Number of Cases	Nationality	Number of Cases	Nationality	Number of Cases
American	2,800	Slav	35	Syrian	8
Italian	463	Bohemian	27	Roumanian	7
German	326	Scotch	27	Welsh	5
Russian	287	Dutch	23	Hindu	5
Polish	211	French	21	Porto Rican	5
Austrian	160	Danish	18	Serbian	3
Irish	128	Lithuanian	18	Belgian	2
English	107	Czechoslovakian	18	Indian	2
Negro	71	Jewish	13	Turk	2
Swedish	66	Greek	13	Portuguese	1
Austro-Hungarian	54	Croatian	12	Filipino	1
Norwegian	50	Swiss	10	Mexican	1
Finnish	35	Spanish	10	Unknown	294
Total					5,348

this report, in spite of the fact that in acquiring data covering the facts in the etiology of deafness there are plenty of opportunities for error. This will be apparent when we point out that the data on this subject had to be gleaned largely from institutional records. We had, of course, no opportunity of visiting the homes of these children, and even when it was possible to talk with the parents it was often impossible to get an accurate knowledge of just what disease the child was suffering from when the deafness came on. In spite of palpable discrepancies and errors which cannot be avoided, I look on the study of the etiology of acquired deafness as most important and illuminating.

I shall not undertake to analyze all the statistics in table 4, but shall content myself with pointing out a few of the outstanding facts. Meningitis heads the list. Deafness developed as a result of this disease in 385, or approximately 19 per cent of the total number of children

TABLE 4.—*Etiology of Acquired Deafness*

Cause	Number	Per Cent	With Otitis Media						Without Otitis Media					
			Total Deafness Vestibular Response			Partial Deafness Vestibular Response			Total Deafness Vestibular Response			Partial Deafness Vestibular Response		
			Normal	Diminished	Absent	Normal	Diminished	Absent	Normal	Diminished	Absent	Normal	Diminished	Absent
Meningitis	385	19.00	1 not tested	1	9	2	1	2	7	43	160	12	50	88
Measles	172	8.54	1	8	5	12	20	5	6	14	22	24	37	18
Scarlet fever	139	6.90	1	5	18	14	15	5	7	10	24	12	25	3
Influenza	139	6.90	1	2	3	5	9	3	9	18	21	20	30	7
														11 not tested
Pneumonia	103	5.11	1	1	2	3	5	..	4	12	27	9	27	12
Suppurative otitis media	87	4.31	6	9	10	30	25	7	..	..	..	..	..	..
Whooping cough	81	3.51	..	1	2	3	4	1	4	6	11	11	23	15
Fall	70	3.47	..	..	1	1	2	1	..	8	6	14	27	10
Infantile paralysis	59	2.93	4 not tested	..	..	2	..	4	3	9	11	3	11	12
Fever (?)	51	2.55	..	1	2	2	1	..	4	6	17	3	11	4
Suppurative otitis media, mastoiditis and operation	47	2.33	1	3	8	13	17	5	..	..	..	..	..	..
Mixed infection	44	2.18	..	..	2	6	6	6	2	2	6	3	7	4
Diphtheria	35	1.73	..	1	1	3	3	..	1	3	7	5	9	2
Typhoid fever	32	1.59	..	..	2	..	2	..	..	3	10	3	6	6
Skull fracture	30	1.49	..	..	..	..	1	..	3	6	12	3	3	2
Probable syphilis	27	1.34	12 not classified			..	..	..	..	..	6	..	1	7
														1 not tested
Brain fever	26	1.33	..	..	5	1	4	..	2	1	1	..	9	3
Mumps	22	1.09	1	2	1	..	1	..	..	2	3	4	4	4
Spasms	18	0.882	1	..	..	..	..	..	..	4	3	2	5	3
Probable otosclerosis	6	0.294	..	..	..	..	..	..	1	..	..	2	3	..
Accidents	4	0.196	..	..	..	..	..	..	..	..	..	1	3	..
Chickenpox	3	0.147	.	..	..	..	..	..	..	1	1	1	..	..
Fright	3	0.147	..	..	..	..	..	..	..	1	..	..	2	..
Colitis	3	0.147	.	..	..	..	..	..	..	.	..	3	..	..
Rickets	2	0.098	.	..	1	..	..	..	..	..	..	1	..	..
Sleeping sickness	1	0.049	..	..	..	..	..	..	..	..	..	..	1	..
Cholera infantum	1	0.049	..	..	..	..	..	..	..	..	..	..	1	..
Stroke lightning	1	0.049	..	..	..	..	..	..	..	..	..	1	..	..
Quinine	1	0.049	..	..	..	..	..	..	..	..	..	1	..	..
Burns	1	0.049	..	..	..	..	..	..	..	..	..	..	..	1
Tuberculosis	1	0.049	..	..	..	..	..	..	..	..	..	..	1	..
Unknown	420	22.10	5	14	9	13	37	11	14	58	51	58	99	44
														7 not tested
Total	2,014	100.00	18	48	81	110	153	50	67	207	408	196	395	245
			5 not tested											19 not tested

examined. The presumption is that although meningitis may develop from several causes, epidemic cerebrospinal meningitis is responsible for a large percentage of these cases. Analyzing the cases of meningitis, we find that 16 were associated with suppurative otitis media and that 369 developed without suppuration of the middle ear. Of the 16 cases associated with otitis media, 11 showed total and 5 partial deafness. Of the 11 children with total deafness, the vestibular responses were absent in 9, diminished in 1 and could not be tested in 1. Of the 5 with partial deafness, vestibular responses were normal in 2, diminished in 1 and absent in 2. Of somewhat more interest are the 369 children who were deaf from meningitis but who had not had an associated otitis media. Of this number, 219 were totally deaf and 150 had partial deafness. The vestibular responses in the 219 cases of total deafness were as follows: In 7 these responses were normal, in 43 they were present but diminished, and in 169 they were absent. Of the 150 children in this group with partial deafness, 12 showed normal vestibular responses, 50 present but diminished responses and 88 no responses.

These statistics covering the cases of deafness resulting from meningitis are particularly illuminating. In a large number in which deafness was only partial, we anticipated total destruction of the hearing function as meningitis had involved the internal ear. Another surprise found in these statistics was the status of the vestibular mechanism. There has been a general impression that when there is acquired deafness—deafness severe enough to necessitate the child's being placed in an institution for the deaf, which means, of course, deafness due to involvement of the labyrinth—the vestibular mechanism suffers to the same extent as the hearing mechanism; in other words, when a child has total deafness that has been acquired there is also a total destruction of the vestibular apparatus. In these statistics on deafness from meningitis, of the 219 children suffering from total deafness 7 showed normal vestibular responses, 43 gave diminished vestibular responses and in 169 the vestibular responses were absent; whereas, of the 150 children with partial deafness vestibular responses were normal in 12, diminished in 50 and absent in 88. It is seen, therefore, that there was no great difference in the status of the vestibular mechanism between the cases of total deafness and those in which the deafness was partial.

The statistics covering the children with deafness acquired from scarlet fever and measles is of great interest, since of all the diseases of childhood these two menace the hearing most often. Whereas, our previous report showed that there were 100 children who had acquired

their deafness through scarlet fever and 97 through measles, our present studies showed only 39 cases resulting from scarlet fever and 75 from measles. In our total statistics 172 children, or 8.54 per cent, acquired deafness from measles and 139, or 6.9 per cent, from scarlet fever. The statistics showing the relation to otitis media in these cases are particularly interesting because of the great frequency of suppuration of the middle ear in both scarlet fever and measles. Of the 139 children suffering from deafness because of scarlet fever, only 58 were found to have suffered from otitis media, while in 81 the deafness developed independent of disease of the middle ear. Of the 172 cases of deafness from measles, 51 were associated with otitis media and 121 developed indepenedent of disease of the middle ear. It is apparent from these figures that the great menace to the hearing in both scarlet fever and measles comes not so much from otitis media as from a toxic neuritis of the eighth nerve. These statistics, of course, do not show the extent of the havoc that both scarlet fever and measles play with the hearing, because suppurative otitis media which involves only the middle ear, in other words, which has not invaded the labyrinth and produced what we term panotitis, does not produce that severe degree of deafness which necessitates the child being placed in an institution for the deaf. A great many persons go through life suffering from partial deafness that is produced by suppurative otitis media which developed in the course of scarlet fever or measles. On the other hand, in view of the large number of cases of severe deafness that occur in both scarlet fever and measles independent of otitis media, it is reasonable to assume that even in cases in which severe deafness has developed in connection with otitis media it is perhaps the associated toxic neuritis of the eighth nerve that is responsible for the severe defect, and that it may be only the exceptional case in which labyrinthine deafness is the result of the invasion of the internal ear by the suppurative disease.

The situation in connection with scarlet fever and measles is of so much interest that I venture to analyze the statistics in this connection shown in table 4. Of the 58 children suffering from deafness acquired during scarlet fever and in whom there was otitis media, 24 were totally and 34 partially deaf. Of the 24 with total deafness, vestibular responses were normal in 1, diminished in 5 and absent in 18. I feel that it is safe to conclude that among these 18 with total deafness and total destruction of the vestibular mechanism are to be found all the cases in which we are justified in assuming that the involvement of the labyrinth was brought about by an invasion of the labyrinth from the suppurative otitis media. To me it seems unthinkable that a suppurative otitis media could invade the labyrinth and still leave any vestige of hearing or of function of the vestibular mechanism. Of

the 34 children with partial deafness acquired from scarlet fever in whom there was an associated otitis media, 14 gave normal vestibular responses; in 15 these responses were present but diminished, and in only 5 were they absent entirely. I am of the impression that in all of these 34 cases the nerve involvement came through toxic neuritis which developed independent of the otitis media.

In regard to the statistics on the 81 cases in the scarlet fever group which developed without otitis media, 41 of these children were totally deaf; yet of this number there were 7 with normal vestibular responses and 10 more in whom the responses were present but diminished, with 24 showing a complete absence of the vestibular responses. Table 4 shows the statistics on cases with or without a history of otitis media, on total and partial deafness and on the relations to vestibular responses similar to those pointed out in the scarlet fever group.

I cannot pass over table 4 without some reference to the cases in which deafness was probably acquired through syphilis. Only 27 cases, or 1.34 per cent, were attributed to probable syphilis. This conclusion, however, was not based on sufficient data, such as the Wassermann test, to make the figures accurate. In the examinations for our previous report all the children in one institution were subjected to the Wassermann test, with the result that of the 396 pupils examined in this way only 2 gave positive Wassermann reactions of the blood. As we pointed out in our previous report, hereditary syphilis does not appear to play an important part in the etiology of acquired deafness among the children in our public institutions for the deaf.

In 30 of the children skull fracture was given as the cause of deafness. This meant that the fracture had passed through both sides of the base of the skull, because if there had been unilateral fracture involving only one ear the children would not have been placed in an institution for the deaf. An analysis of the statistics in these 30 cases is of considerable interest for the reason that when deafness results from a fracture through the base of the skull the injury to the labyrinth presumably would result in the total destruction not only of the hearing, but of the vestibular mechanism as well. Only 1 of the 30 cases developed in connection with otitis media. One should not infer from this figure that otitis media does not frequently complicate a fracture at the base of the skull. As a matter of fact, a fracture at the base of the skull not infrequently lacerates the membrana tympani, and when this takes place otitis media is prone to develop, especially if some one undertakes to cleanse the external meatus of blood by irrigation. When otitis media develops in connection with a fracture at the base of the skull, fatal meningitis is prone to supervene. Of the 29 cases of frac-

ture of the skull without otitis media, there was total deafness in 21, and partial deafness in 8. The latter figure seems rather large, and may be accounted for, at least partially, as a result of error, the parent attributing to a fall a defect in hearing which he is loathe to accept as congenital. If the statistics are taken at their face value, however, the 21 children with total deafness included 3 with normal vestibular responses, 6 with diminished responses and 12 with these responses entirely absent. Whereas of the remaining 8 children with deafness attributed to fracture of the skull, vestibular responses were normal in 3, diminished in 3 and absent in 2. In connection with the statistics on deafness acquired from skull fracture, I would call attention to the fact that in the same table there are 70 cases of deafness reported as resulting from a fall. Since the only way in which a fall can produce deafness is by fracture of the skull, it seems probable that most of the cases reported as resulting from a fall were cases in which the parents were anxious to find some cause for acquired deafness; when no illness occurred, the theory of a fall was resorted to, perhaps because every infant has more than one serious fall which the parents could accept as a cause of deafness.

MUMPS

Mumps plays its rôle in destruction of the hearing. In 22 of the children this was given as the cause, and this figure can be taken as accurate, for mumps is readily recognized, and the defect in hearing comes suddenly. In 17 of these children the involvement of the labyrinth was not associated with otitis media. Total deafness was found in only 5; in 12 the defect in hearing was only partial. Here again we are interested particularly in the status of the vestibular mechanism. In 2 of the children with total deafness, vestibular responses were present but diminished; that is, the nystagmus after rotation was less than 15 seconds. In the remaining 3 no response was elicited by rotation. Of the 12 children with partial deafness, 4 showed normal vestibular responses, 4 diminished responses and 4 absence of responses. It is apparent, therefore, that in this involvement of the labyrinth caused by mumps, the hearing is sometimes totally lost and that in others the defect is only partial; the vestibular apparatus may be partially or completely destroyed or may be left intact.

THE RELATION OF TONSILS AND ADENOIDS TO LESIONS OF THE MIDDLE EAR

Among the children examined, 2,355, or 44.03 per cent, had their tonsils and adenoids removed, 2,638, or 49.32 per cent, still had their tonsils and of 355 no record was made regarding the tonsils. Of the

children who had the tonsils and adenoids removed, 1,377 showed no palpable evidence of having had middle ear trouble. Of the children with tonsils still in situ, 1,062 showed palpable evidence of middle ear disease and in 1,576 such evidence was absent. Tonsil and adenoid trouble can be responsible for severe deafness only when it has produced otitis media which has invaded the labyrinth—a negligible factor in these statistics.

TABLE 5.—*Relation of Tonsil and Adenoid Operations to Lesions of the Middle Ear, Total Deafness and Partial Deafness*

Number of children who had tonsils and adenoids successfully removed 2,355 (44.03%)	No. of cases with palpable evidence of lesion of middle ear 978 (41.53%)	Suppuration 283 (28.93%)	Total deafness 99 (34.83%)
			Partial deafness 184 (65.17%)
		Tubotympanic catarrh 695 (71.07%)	Total deafness 249 (39.12%)
			Partial deafness 446 (63.88%)
	No. of cases without palpable evidence of lesion of middle ear..... 1,377 (58.47%)		Total deafness 479 (34.78%)
			Partial deafness 898 (65.22%)
No record, 355 (6.65%)			
Number of children with tonsils still in situ 2,638 (49.32%)	No. of cases with palpable evidence of lesion of middle ear 1,062 (40.26%)	Suppuration 215 (20.24%)	Total deafness 85 (39.53%)
			Partial deafness 130 (60.47%)
		Tubotympanic catarrh 847 (79.76%)	Total deafness 378 (44.62%)
			Partial deafness 469 (55.38%)
	No. of cases without palpable evidence of lesion of middle ear..... 1,576 (59.74%)		Total deafness 680 (43.07%)
			Partial deafness 896 (56.93%)

HEREDITY AND CONSANGUINITY

Table 6 shows the relation of heredity and consanguinity in 1,374 children, a fact which in itself shows clearly the danger of intermarriage. In 112 cases the parents were first cousins; in 64 the parents were second, in 36 third and in 18 fourth cousins. This table does not bring out the interesting question as to the extent to which deafness was found to be hereditary in the parents of the children. A study of the reports of the individual cases brings out this fact. The table does show the hereditary factor of deafness in the families of the parents. In 112 instances there were deaf uncles, in 126 instances deaf aunts and in 175 instances deaf cousins. In 144 cases parents were deaf, in 40 cases grandparents and in 59 cases other relatives. The table showing deafness in the immediate family, that is, the number of other children deaf, is of interest. In 484 cases, another child in the same family was deaf, in 211 two others, in 47 three others, in 13 four others and in 6 five others; 17 pairs of twins were deaf.

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children*

Summary

Number of cases		1,374
Nature of consanguinity	First cousins	112
	Second cousins	64
	Third cousins	36
	Fourth cousins	18
	Uncle and niece	1
	Father and daughter	1
Deafness in family	Parents	144
	Uncles	112
	Aunts	126
	Cousins	175
	Grandparents	40
	Others	59
Deafness in immediate family — Number other children deaf	1 other	484
	2 others	211
	3 others	47
	4 others	13
	5 others	6
	Twins	17 pairs
Deafness	Total	511
	Partial	854
	No record	9
Vestibular response	Normal	445
	Diminished	668
	Absent	252
	No record	5

*Observations in Individual Cases**

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
724. Annie T., 5 yrs.; vestige of hearing; no vestibular response	None	None	One brother
725. Josephine J., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
726. Josephine S., 7 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
727. Millie C., 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
728. Doris R., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
729. Raymond R., 5 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
730. Vincent A., 9 yrs.; vestige of hearing; no vestibular response	None	Parents	None
731. Warrian C., 6 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother

* The first 723 cases in this study were reported in Shambaugh, Hagens, Holderman and Watkins: Statistical Studies of the Children in the Public Schools for the Deaf, Arch. Otolaryng. **7**: 424 (May) 1928.

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
732. Kenneth F., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
733. Patsy C., 15 yrs.; no vestige of hearing; no vestibular response	None	None	One sister
734. Russell S., 19 yrs.; vestige of hearing; no vestibular response	None	Several cases on mother's side	None
735. Raymond P., 15 yrs.; vestige of hearing; vestibular responses diminished	None	Parents	None
736. Martin L., 17 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
737. Lester B., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister; one brother
738. George S., 16 yrs.; no vestige of hearing; vestibular responses normal	None	None	Two brothers
739. William S., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
740. Seybert Van., 15 yrs.; vestige of hearing; vestibular responses diminished	None	Cousin	One brother; one sister
741. John Van., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	Cousin	One brother; one sister
742. Edward S., 19 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
743. Michael H., 19 yrs.; no vestige of hearing; no vestibular response	None	None	One sister
744. Seymour S., 16 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
745. John D., 14 yrs.; no vestige of hearing; vestibular responses normal	None	Parents	None
746. Charles M., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	Two sisters
747. Walter P., 13 yrs.; vestige of hearing; vestibular responses normal	None	Father, mother and uncle	None
748. Albert L., 10 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
749. Helen W., 6 yrs.; vestige of hearing; no vestibular response	None	None	One sister
750. Florence M., 5 yrs.; no vestige of hearing; vestibular responses diminished	None	Uncle and others None	None
751. Anna B., 5 yrs.; vestige of hearing; no vestibular response	None	None	One sister; one brother
752. Catherine W., 10 yrs.; vestige of hearing; vestibular response diminished	None	None	One sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
753. Catherine C., 11 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
754. Edith D., 7 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
755. Edith Dun., 9 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Two brothers
756. Anita W., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
757. Emil O., 10 yrs.; vestige of hearing; no vestibular response	None	None	Four sisters (twin)
758. Frances O., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	Four sisters (twin)
759. Mary S., 15 yrs.; no vestige of hearing; vestibular desponses diminished	None	None	One brother
760. Ruth M. L., 10 yrs.; vestige of hearing; no vestibular response	None	None	One sister
761. Filomena D. L., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister; one brother
762. Dorothy E. B., 10 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
763. Jeanette W., 17 yrs.; no vestige of hearing; vestibular responses normal	None	One cousin	None
764. Helen M., 14 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister; one brother
765. Loretta K., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
766. Anna O., ? yrs.; vestige of hearing; vestibular responses diminished	None	None	Four sisters
767. Sadie O., 16 yrs.; no vestige of hearing; vestibular responses normal	None	None	Four sisters
768. Agnes M., 18 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother; one sister
769. Elizabeth L., 19 yrs.; vestige of hearing; vestibular responses diminished	None	None	Grandmother
770. Rosie V., 11 yrs.; vestige of hearing; vestibular responses diminished	Third cousin	None	None
771. Esther D., 18 yrs.; vestige of hearing; vestibular responses diminished	Second cousin	None	One sister
772. Carol D., 14 yrs.; vestige of hearing; vestibular responses normal	Second cousin	None	One sister
773. Emma Van D. H., 11 yrs.; vestige of hearing; no vestibular response	None	One cousin	Two brothers

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
774. Joe C., 17 yrs.; vestige of hearing; no vestibular response	Not stated	None	None
775. Fannie K., 17 yrs.; no vestige of hearing; vestibular responses normal	First cousin	None	None
776. John R., 7 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
777. George P., 6 yrs.; no vestige of hearing; no vestibular response	None	Father and mother	None
778. Charles W., 7 yrs.; no vestige of hearing; vestibular response diminished	None	None	Two brothers
779. George B., 7 yrs.; no vestige of hearing; vestibular response diminished	None	Father and mother	None
780. Thomas C., 7 yrs.; no vestige of hearing; vestibular response diminished	None	Father	None
781. Mike G., 11 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother
782. Bernard C., 9 yrs.; no vestige of hearing; vestibular responses	None	None	One sister
783. Frank G., 13 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
784. Tony Sch., 10 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
785. Peter V., 8 yrs.; vestige of hearing; no vestibular response	None	Three deaf cousins	None
786. Norman D., 9 yrs.; vestige of hearing; no vestibular response	None	None	One sister
787. Vincent S., 9 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
788. Robert Bl., 9 yrs.; vestige of hearing; no vestibular response	None	None	One brother; one sister
789. Allen P., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father, mother and uncle	One brother
790. Frank R., 13 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
791. Herbert S., ? yrs.; vestige of hearing; no vestibular response	None	None	One brother
792. Margaret S., 18 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
793. Melba C., 15 yrs.; vestige of hearing; no vestibular response	None	None	One brother
794. Florence L., 12 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
795. Myrtle B., 11 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother
796. Irma L., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
797. Amos F., 17 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother
798. Helen H., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
799. Dorothy C., 15 yrs.; no test	None	None	Deaf brothers and sisters
800. Violet Bl., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
801. Sophie W., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
802. Clara T., 15 yrs.; vestige of hearing; no vestibular response	Cousins	None	Four brothers; four sisters
803. Vincent C., 7 yrs.; vestige of hearing; no vestibular response	First cousins	None	None
804. Michael G., 12 yrs.; vestige of hearing; no vestibular response	Brother and Sister	None	None
805. Peter Tr., 15 yrs.; vestige of hearing; vestibular responses normal	Cousins	None	None
806. Fred S., 13 yrs.; no vestige of hearing; no vestibular responses	Cousins	Grandmother	None
807. Georgia B., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
808. Haywood W., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
809. Charles W., 11 yrs.; vestige of hearing; no vestibular response	Cousins	None	One brother
810. Harry W., 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
811. Charles F., 11 yrs.; vestige of hearing; vestibular responses normal	None	Distant relatives	None
812. Wilson, Sh., 18 yrs.; vestige of hearing; vestibular responses normal	None	Relatives	Father; mother; two brothers; two sisters
813. Ray S. H., 20 yrs.; no vestige of hearing; vestibular responses normal	None	Relatives	Father; mother; two brothers; two sisters
814. John S., 11 yrs.; vestige of hearing; vestibular responses normal	None	Cousins	
815. Lillie McC., ? yrs.; no vestige of hearing; vestibular responses diminished	Cousins	Mother	One sister
816. Floyd M., 13 yrs.; vestige of hearing; vestibular responses normal	Cousins	Relative	One sister
817. Rosa Mc., 13 yrs.; no vestige of hearing; vestibular responses diminished	Cousins	Mother	One sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
818. Beulah S., 7 yrs.; no vestige of hearing; vestibular responses normal	Third cousins	None	None
819. Elma L., 15 yrs.; no vestige of hearing; vestibular responses diminished	Second cousins	None	None
820. Mary C., 10 yrs.; vestibular responses normal	First cousins	None	None
821. Pennie M., 14 yrs.; no vestige of hearing; vestibular responses diminished	Third cousins	None	None
822. Leonard A., 9 yrs.; no vestige of hearing; no vestibular response	First cousins	None	None
823. Roy Ch., 15 yrs.; no vestige of hearing; vestibular responses diminished	Cousins	None	None
824. Maude S., 14 yrs.; no vestige of hearing; vestibular responses diminished	Cousins		One sister
825. Margaret H., 18 yrs.; no vestige of hearing; vestibular responses diminished	Second cousins	None	None
826. Eugene F., 10 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	One brother
827. Don F., 12 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	One brother
828. Sandy C., 13 yrs.; vestige of hearing; vestibular responses diminished	Third cousins	None	One brother
829. Alden B., 16 yrs.; no vestige of hearing; no vestibular response	Cousins	None	None
830. Zeb. A., 8 yrs.; no vestige of hearing; vestibular responses diminished	Fourth cousins	None	One sister
831. Jarvis C., 18 yrs.; vestige of hearing; vestibular responses normal	Third cousins	None	One brother
832. Annie B., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers
833. Nina W., 19 yrs.; vestige of hearing; vestibular responses normal	None	Grandmother	None
834. Norma R., 14 yrs.; vestige of hearing; vestibular responses diminished	Third cousins	None	None
835. Bernice B., 15 yrs.; no vestibular responses	First cousins	None	Two sisters
836. Jeanette A., 12 yrs.; vestige of hearing; vestibular responses diminished	Fourth cousins	None	One brother; one sister
837. Paul L., 12 yrs.; vestige of hearing; vestibular responses diminished	Third cousins	None	None
838. Wilson M., 10 yrs.; vestige of hearing; vestibular responses diminished	Distant cousins	None	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
839. Joe M., 10 yrs.; vestige of hearing; vestibular responses normal	Distant cousins	None	None
840. John H., 14 yrs.; vestige of hearing; vestibular responses normal; twin of following patient	Fourth cousins	None	One brother; one sister
841. James H., 14 yrs.; no vestige of hearing	Fourth cousins	None	One brother; one sister
842. Spencer H., 11 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	None
843. Luther Y., 10 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Two sisters
844. Gerald W., 7 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Two sisters
845. Pershing S., 11 yrs.; vestige of hearing; vestibular responses diminished	None	Father, mother and cousins	One sister
846. Ollie S., 12 yrs.; no vestige of hearing; no vestibular response	None	Father	One brother; one sister
847. Allen Sh., 10 yrs.; vestige of hearing; no vestibular response	None	Father and grandmother	One brother; one sister
848. Reid P., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	One brother; one sister
849. Kramer P., 8 yrs.; vestige of hearing; no vestibular response	None	"Five cases of deafness in family"	
850. Tyson P., 15 yrs.; no vestige of hearing; vestibular responses normal	None	Third cousins and fourth cousins	None
851. Jack M., 7 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
852. Oscar S., 15 yrs.; no vestige of hearing; vestibular responses diminished	None	Two aunts	None
853. Howard H., 15 yrs.; no vestige of hearing; vestibular responses normal	None	One third cousin	None
854. Louise W., 15 yrs.; vestige of hearing; vestibular responses diminished	None	Second cousin	None
855. Maxine W., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
956. Francis M., 13 yrs.; no vestige of hearing; vestibular responses diminished	None	"All through family"	None
857. Carrie M., 10 yrs.; no vestige of hearing; vestibular responses normal	None	Father, mother and "others"	None
858. Grover P., 18 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One sister
859. Ira M., 19 yrs.; vestige of hearing; vestibular responses diminished	None	Two uncles and one aunt	One sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
860. Hugh H., 12 yrs.; no vestige of hearing; no vestibular response	None	Several cousins	None
861. Clara Y., 8 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	One brother; one sister
862. Virgie T., 9 yrs.; no vestige of hearing; vestibular responses normal	None	Two third cousins	None
863. Cora M., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
864. Lunetta L., 10 yrs.; no vestige of hearing; vestibular responses diminished	None	Cousins	None
865. Louise D., 10 yrs.; vestibular responses diminished	None	None	One sister
866. Mildred D., 8 yrs.; vestige of hearing; no vestibular response	None	Two second cousins	None
867. Bettie S., 16 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Three brothers; one sister
868. Wauline S., 15 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
869. Mamie S., 19 yrs.; vestige of hearing; no vestibular response	None	None	One sister
870. Nelda R., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father, mother, cousins and uncle	None
871. Virginia P., 11 yrs.; no vestige of hearing; no vestibular response	None	Father and mother	One brother
872. Virginia J., 8 yrs.; vestige of hearing; vestibular responses diminished	None	Grandmother and uncle	None
873. Fannie H., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
874. Flossie D., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother; one sister
875. Mildred D., 8 yrs.; no vestige of hearing; no vestibular response	None	Second cousin	None
876. Coltie D., 17 yrs.; vestige of hearing; vestibular responses normal	None	Cousins	None
877. Annie M. C., 14 yrs.; vestige of hearing; vestibular responses diminished	None	One cousin	None
878. Muriel C., 15 yrs.; no vestige of hearing; vestibular responses normal	None	None	Two sisters
879. Dorothy C., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
880. Cassie B., 9 yrs.; vestige of hearing; vestibular responses normal	None	Mother and three others	None
881. Styles, P., 11 yrs.; vestige of hearing; no vestibular response	None	Father and mother	Brother and sisters

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
882. Broadie, P., 10 yrs.; vestige of hearing; vestibular responses normal	None	One cousin	None
883. Edgar N., 10 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
884. Mack M., 10 yrs.; no vestige of hearing; vestibular responses normal	None	One (?)	None
885. Tate, L., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
886. Carrie J., 10 yrs.; vestige of hearing; no vestibular response	None	Father; grandfather; three uncles; many cousins	Two brothers; one sister
887. Clarence H., 20 yrs.; vestige of hearing; vestibular responses normal	None	Second cousins	None
888. Lawrence G., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Grandfather	None
889. Morris H., 11 yrs.; vestige of hearing; vestibular responses normal	None	Uncle	None
890. Melvine D., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
891. Ralph C., 9 yrs.; vestige of hearing; vestibular responses normal; twin of following patient	None	Father and mother	Three brothers
892. Paul C., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	Three brothers
893. John C., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Three brothers
894. Edgar C., 14 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	Three brothers
895. Seldon C., 13 yrs.; vestige of hearing; no vestibular response	None	None	One brother; one sister
896. Durwood B., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Cousins	None
897. Ralph A., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Cousin	None
898. Ray G., 18 yrs.; vestige of hearing; vestibular responses normal	None	Father and grandmother	None
899. George H., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	Twin brother
900. Oscar B., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister
901. Michael Del V., 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
902. Tony M., 14 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
903. Stanley Z., 20½ yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
904. Elias S., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister
905. Edward Z., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
906. John M., 14 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	One brother
907. Frank M., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother; one sister
908. Chester G., 14 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	None
909. Hudnel L., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
910. Ernest S., 11 yrs.; vestige of hearing; vestibular responses normal	None	Father; mother; father's brother; mother's two brothers and two sisters	One sister
911. Ignatz A., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	Mother's brother	None
912. Richard T., 16 yrs.; no vestige of hearing; vestibular responses diminished	None	Mother's brother and cousin	None
913. Hyman W., 13 yrs.; no vestige of hearing; no vestibular response	None	None	Two sisters
914. Joseph Del S., 15 yrs.; no vestige of hearing; no vestibular response	None	None	One sister
915. Harold L., 13 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One brother; one sister
916. Clair J. F., 15 yrs.; vestige of hearing; vestibular responses diminished	None	Father's brother and niece	None
917. Philip T., 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
918. Morton R., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
919. Alphonse S., 16 yrs.; vestige of hearing; vestibular response normal	None	Father's sister	None
920. John G., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
921. Thomas M., 13 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
922. Susie K., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
923. Hilda L., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
924. Maza L., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
925. Jean M., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
926. Sarah H., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
927. Janet M. K., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
928. Ruth I. P., 12 yrs.; vestige of hearing; vestibular responses diminished	None	Two cousins	None
929. Anna B., 13 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
930. Lura H., 13 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
931. Florence K., 12 yrs.; no vestige of hearing; no vestibular response	None	None	One brother
932. Lenora P., 12 yrs.; vestige of hearing; vestibular responses normal	None	One cousin	One sister
933. Irene W., 14 yrs.; vestige of hearing;; vestibular responses normal	None	Father's two nephews; mother's uncle and aunt	Two brothers
934. Fanny W., ? yrs.; no vestige of hearing; no vestibular response	None	None	One brother; one sister
935. Dinah R., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother
936. Veronica O., 16 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; two sisters
937. Louise W., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Mother's aunt	One sister
938. Helen C. P., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
939. Dorothy W., 13 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None
940. Wilda C., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	Maternal grandmother and great grandmother	None
941. Evelena Del S., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
942. Phyllis L. P., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
943. Doris A. S., 15 yrs.; vestige of hearing; vestibular responses diminished	None	One cousin	None
944. Josephine R., 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
945. Nettie M., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
946. Rose Z., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
947. Charles E. B., 12 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One sister
948. Walter F. R., 11 yrs.; vestige of hearing; vestibular responses normal	None	Father's great aunt and uncle	None
949. Irene E., 12 yrs.; vestige of hearing; vestibular response normal	Second cousins	None	One sister
950. Nicholas P., 12 yrs.; vestige of hearing; no vestibular response	None	First cousins	None
951. Robert Y., 22 yrs.; no vestige of hearing; vestibular responses diminished	Second cousins	None	None
952. Andrew T., 12 yrs.; no vestige of hearing; vestibular responses diminished	None	Parents born deaf	One half-brother
953. Rocco M., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
954. Earl M., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
955. Fred S., 7 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
956. Gilbert McN., 15 yrs.; vestige of hearing; vestibular responses diminished	None	One maternal uncle	One brother
957. Clement F., 16 yrs.; vestige of hearing; no vestibular response	None	One uncle; one aunt	None
958. Ruth W., 16 yrs.; vestige of hearing; vestibular responses diminished	None	One great aunt; one uncle	One brother; one sister
959. Marian P., 14 yrs.; vestige of hearing; no vestibular response	None	None	One sister
960. Martha B., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
961. Wesley F., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
962. Ralph W., 17 yrs.; no vestige of hearing; no vestibular response	None	Mother hard of hearing	Two sisters
963. Stanley B., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
964. Elmer W., 16 yrs.; vestige of hearing; vestibular responses diminished	None	Two cousins; one great aunt; one great uncle	One brother; one sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
965. Helen R., 18 yrs.; vestige of hearing; no vestibular response	None	None	One brother
966. Grace B., ? yrs.; no vestige of hearing; no vestibular response	None	Mother's cousin partially deaf, with defective auricles	None
967. Allen C., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	One uncle	None
968. Mary M., 14 yrs.; vestige of hearing; no vestibular response	None	None	Three brothers; one sister
969. Jacob K., ? yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
970. Etmere A., 16 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	Three brothers; one sister
971. Bud F., 19 yrs.; vestige of hearing; vestibular responses diminished	None	Maternal grandmother	One brother
972. Leah S., 17 yrs.; no vestige of hearing; no vestibular response	None	Great grandfather; two great uncles; two great aunts; one second cousin; all maternal relations	None
973. Elsie Z., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
974. Helen K., 19 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
975. Amelia N., 16 yrs.; vestige of hearing; vestibular responses diminished	None	Grandmother	None
976. Ester H., 20 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
977. Christian N., 18 yrs.; vestige of hearing; vestibular responses normal	None	Aunt	None
978. Abraham W., 20 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
979. Grace H., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
980. Myrtle L., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Parents; two aunts	Two brothers
981. Jerry K., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
982. Philip Z., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
983. Warren H., ? yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
984. Esther P., 14 yrs.; no vestige of hearing; no vestibular response	Yes — degree not given	None	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
985. Samuel P., 18 yrs.; vestige of hearing; no vestibular response	First cousins	None	None
986. Steven G., 16 yrs.; vestige of hearing; vestibular responses normal	Second cousins	None	None
Eg. Family:			
987. Helen, 14 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
988. John, 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
989. Frank A., 15 yrs.; vestige of hearing; no vestibular response	None	None	Two brothers
999. Goldie B., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1000. Mike B., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1001. Thomas C., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Parents of mother; father; mother	None
1002. Elmer C., 14 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	None
1003. Paul C., 17 yrs.; vestige of hearing; no vestibular response	None	None	One sister
1004. Susan D., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1005. Alice F., 8 yrs.; vestige of hearing; vestibular responses diminished	None	Brother of mother; father; mother	None
1006. Frank F., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1007. Jake G., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None
1008. Emma G., 7 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
1009. Cyril H., 11 yrs.; vestige of hearing; no vestibular response	None	Two cousins	One brother
1010. Elizabeth H., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1011. Frank H., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1012. James J., 6 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1013. Marby L., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1014. John M., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1015. Martin I., 6 yrs.; vestige of hearing; no vestibular response	None	Sister of father	None
1016. Paul O., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	Three sisters
1017. Anna P., 10 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One sister
1017. Joseph P., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father; two sisters of father	One sister
1018. Alice R., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1019. Robert S., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1020. Donald S., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1021. Mary K., 7 yrs.; vestige of hearing; no vestibular response	None	None	One sister
1022. Armand M., 8 yrs.; vestige of hearing; no vestibular response	None	None	One brother
1023. Virginia R., 6 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1024. Ruth S., 8 yrs.; vestige of hearing; no vestibular response	None	Father; mother; two brothers and two sisters of mother	One brother
1025. Elaine S., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1026. Raymond W., 8 yrs.; vestige of hearing; vestibular responses diminished	None	Two nephews of father	One brother; one sister
1027. Martin Z., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
B. Family:	None	None	One brother (no. 1029); one sister
1028. Ruth, 8 yrs.; no vestige of hearing; vestibular responses normal			
1029. Ramon, 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
C. Family:			
1030. Gladys, 9 yrs.; no vestige of hearing; vestibular responses diminished	None	Two cousins (twins)	One sister
1031. Elizabeth, 10 yrs.; vestige of hearing; vestibular responses normal	None	Two cousins (twins)	One sister
Cr. Family:			
1032. Lorraine, 6 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
1033. Gerald, 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
D. Family:			
1034. Sophia, 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1035. Ladislaw, 10 yrs.; vestige of hearing; no vestibular response	None	None	One sister
E. Family:			
1036. Florence, 7 yrs.; no vestige of hearing; vestibular responses diminished	None	None	Two brothers
1037. John, 6 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
O. Family:			
1038. Jacob, 6 yrs.; no vestige of hearing; no vestibular response	None	None	One sister
1039. Mildred, 10 yrs.; vestige of hearing; no vestibular response	None	None	One brother
P. Family:			
1040. Mildred, 8 yrs.; vestige of hearing; no vestibular response	None	None	One brother
1041. Alexander, 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
Sen. Family:			
1042. John, 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1043. Henry, 11 yrs.; vestige of hearing; vestibular responses diminished; twin of following patient	None	None	One brother; one sister
1044. Henrietta, 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
Sh. Family:			
1045. Doris, 9 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
1046. Dick, 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
Sw. Family:			
1047. Anne, 9 yrs.; vestige of hearing; no vestibular response	None	None	One brother; one sister
1048. Helen, 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
Za. Family:			
1049. Fred, 8 yrs.; vestige of hearing; vestibular responses diminished	None	Two cousins	One sister
1050. Joseph, 6 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
Zu. Family:			
1051. Joseph, 9 yrs.; vestige of hearing; no vestibular response	None	None	One brother; one sister
1052. Bennie, 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1053. Arthur A., 9 yrs.; vestige of hearing; vestibular responses normal	None	Aunt	One sister
1054. Frank A., 9 yrs.; no vestige of hearing; vestibular responses normal	None	Maternal cousins	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1055. Frank A., 17 yrs.; vestige of hearing; vestibular responses normal	None	Father	None
1056. Lois A., 11 yrs.; vestige of hearing; vestibular responses diminished	Third cousins	Mother	One sister
1057. Aumon B., ? yrs.; vestige of hearing; vestibular responses normal	Cousins	None	None
1058. Lizzie B., 18 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	Two cousins	Two brothers
1059. Jimmie B., 10 yrs.; vestige of hearing; vestibular responses diminished	Third cousins	None	One sister
1060. Goldie B., 14 yrs.; no vestige of hearing; no vestibular response	First cousins	None	Two brothers
1061. Bascon B., 13 yrs.; vestige of hearing; no vestibular response	None	One aunt	None
1062. Rosa B., 17 yrs.; vestige of hearing; no vestibular response	Third cousins	None	Two brothers; one sister
1063. Glenn C., 15 yrs.; vestige of hearing; vestibular responses normal	None	Several cousins	None
1064. Nellie C., 16 yrs.; no vestige of hearing; no vestibular response	None	Several cousins	None
1065. Lottie C., 19 yrs.; vestige of hearing; vestibular responses normal	None	Father; mother; grandparents; maternal uncles and cousins	None
1066. Jasper C., 12 yrs.; no vestige of hearing; vestibular responses diminished	None	Mother born deaf	None
1067. Harmon C., 21 yrs.; vestige of hearing; vestibular responses normal	None	Father	One brother
1068. Ethel C., 10 yrs.; vestige of hearing; vestibular responses normal	None	Two aunts and cousins	One brother; one sister
1069. Willie C., 11 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	Two sisters
1070. Robert C., 11 yrs.; no vestige of hearing; vestibular responses diminished	Third cousins	None	None
1071. Clyde C., 14 yrs.; vestige of hearing; vestibular responses normal	None	Father has deaf brother and mother two deaf brothers	None
1072. Ward D., 14 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	None
1073. John W. D., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1074. Hazel F., 8 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	None
1075. Albert F., 18 yrs.; vestige of hearing; vestibular responses diminished	None	Great grandfather	One brother
1076. Alice F., 20 yrs.; vestige of hearing; vestibular responses diminished	None	One aunt	None
1077. Charlotte G., 18 yrs.; vestige of hearing; vestibular responses diminished	First cousins	One cousin	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1078. Pasey G., 10 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	None
1079. Robert G., ? yrs.; vestige of hearing; vestibular responses diminished	None	Father, mother, uncle and aunt	Two sisters
1080. Lucille H., ? yrs.; vestige of hearing; vestibular responses normal	Third cousins	Two aunts; two nephews	None
1081. Oscar H., ? yrs.; vestige of hearing; no vestibular response	Cousins	One maternal uncle and two cousins	One sister
1082. Elizabeth H., 20 yrs.; vestige of hearing; vestibular responses diminished	None	Cousin	One brother
1083. Nannie H., 17 yrs.; no vestige of hearing; vestibular responses diminished	Fourth cousins	None	None
1084. Charles J., ? yrs.; no vestige of hearing; vestibular responses normal	First cousins	Cousins	None
1085. Alma J., 20 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1086. Jessie J., 13 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None
1087. Mary K., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1088. Zella L., 20 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	None
1089. Dorothy L., 16 yrs.; vestige of hearing; too ill to rotate	First cousins	None	One brother
1090. Robert L., 20 yrs.; vestige of hearing; vestibular responses diminished	None	Several cousins	None
1091. Lacie M., 18 yrs.; vestige of hearing; no vestibular response	None	None	One sister
1092. Tiny M., 18 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
1093. Claudie Mc., 16 yrs.; no vestige of hearing; vestibular responses diminished	Third cousins	None	None
1094. Roy M., 9 yrs.; vestige of hearing; no vestibular response	First cousins	Father; cousins	One sister
1095. Preston N., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	None
1096. Margaret O., 9 yrs.; no vestige of hearing; vestibular responses diminished	Third cousins	Father's uncle	None
1097. Earl P., 14 yrs.; vestige of hearing; no vestibular response	None	Father had deaf relatives	None
1098. William P., 20 yrs.; vestige of hearing; vestibular responses diminished	None	Cousins	Two brothers; one sister
1099. David R., ? yrs.; vestige of hearing; vestibular responses normal	Cousins	Father; maternal grandparents	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1100. Louis R., ? yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1101. James R., 17 yrs.; vestige of hearing; vestibular responses diminished	Cousins	Father; maternal grandparents	One brother
1102. Kenneth R., 17 yrs.; vestige of hearing; Vestibular responses normal	None	Two cousins of mother deaf	None
1103. Lela R., 16 yrs.; vestige of hearing; vestibular responses diminished	First cousins	One cousin	One brother
1104. Helen R., 9 yrs.; vestige of hearing; vestibular responses normal	Cousins	Great uncles and aunts	None
1105. Ruth S., 9 yrs.; vestige of hearing; no vestibular response	Cousins	None	None
1106. Frances S., 15 yrs.; vestige of hearing; vestibular responses normal	Second cousins	One uncle	One sister
1107. Margaret S., 21 yrs.; vestige of hearing; vestibular responses diminished	None	Two cousins	None
1108. Billie V., 18 yrs.; vestige of hearing; no vestibular response	Cousins	None	None
1109. Margaret V., 5 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1110. Alma W., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Father; one aunt; one uncle	One brother; one sister
1111. Pansey W., 13 yrs.; vestige of hearing; vestibular responses diminished	None	Cousins	None
1112. Mamie E., 17 yrs.; vestige of hearing; vestibular responses diminished	Fourth cousins	None	None
1113. Bessie W., 19 yrs.; vestige of hearing; vestibular responses diminished	None	Several cousins	None
1114. Mae W., 21 yrs.; vestige of hearing; vestibular responses diminished	Fourth cousins	Four cousins	Two brothers
1115. Mealia Y., 8 yrs.; no vestige of hearing; no vestibular response	Cousins	None	None
Cl. Family:			
1116. Comensia, 17 yrs.; no vestige of hearing; vestibular responses diminished	Fourth cousins	None	One brother
1117. Andrew, 21 yrs.; vestige of hearing; vestibular responses diminished	Fourth cousins	None	One sister
1118. Ida, 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1119. Louis, 20 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
Giff. Family:			
1120. James, 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1121. Mazie, 19 yrs.; vestige of hearing; Vestibular responses normal	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
Garr. Family: (Six children in a family of fourteen were deaf; three of them were examined)			
1122. Dorothy, 15 yrs.; no vestige of hearing; no vestibular response	First cousins	Two cousins	Two sisters; three brothers
1123. Ruby, 8 yrs.; no vestige of hearing; no vestibular response	First cousins	Two cousins	Two sisters; three brothers
1124. Thomas, 10 yrs.; no vestige of hearing; no vestibular response	First cousins	Two cousins	Three sisters; two brothers
Hal. Family:			
1125. Catherine, 11 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One brother; two sisters
1126. Lena, 9 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One brother; two sisters
Kai. Family:			
1127. Ray, 12 yrs.; vestige of hearing; vestibular responses diminished	First cousins	Two cousins	One brother; one sister
1128. Roy, 18 yrs.; vestige of hearing; vestibular responses diminished	First cousins	Two cousins	One brother; one sister
1129. Pearl, 9 yrs.; vestige of hearing; vestibular responses diminished	First cousins	Two cousins	Two brothers
Kai. Family (cousins of preceding Kai. family):			
1130. Ralph, 8 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	Third cousins	One sister
1131. Helen, 12 yrs.; vestige of hearing; vestibular responses normal	Second cousins	Third cousins	One brother
L. Family:			
1132. James, 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1133. Arthur, 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
Oak. Family:			
1134. Mildred, 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1135. Dorothy, 7 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
Pril. Family:			
1136. Bettie, 15 yrs.; vestige of hearing; vestibular responses diminished	None	One maternal uncle; one paternal aunt	One brother
1137. Cecil, 11 yrs.; vestige of hearing; vestibular responses diminished	None	One maternal uncle; one paternal aunt	One sister
Ragl. Family:			
1138. Marie, 12 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One sister
1139. Elsie, 15 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	One sister
1140. Charles S., 16 yrs.; vestige of hearing; vestibular responses diminished	Cousins	Cousins	One brother
1141. Pauline S., 20 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	Several cousins	One sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1142. Ernest M., 19 yrs.; vestige of hearing; vestibular responses normal	None	Father; mother; uncle	None
1143. Albert P., 17 yrs.; vestige of hearing; vestibular responses normal	None	Father; mother; uncle; Grandfather	One brother; one sister
1144. Louise W., 19 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1145. Harry G., 11 yrs.; vestige of hearing; vestibular responses normal	None	Greatuncle	Two brothers
1146. Irving G., 12 yrs.; vestige of hearing; vestibular responses diminished	None	Greatuncle	Two brothers
1147. Frederick N., 12 yrs.; vestige of hearing; vestibular responses diminished	None	Two cousins	None
1148. Jerry D., 12 yrs.; vestige of hearing; no vestibular response	None	None	Two sisters
1149. Josephine S., 12 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1150. Albert B., 16 yrs.; vestige of hearing; vestibular responses diminished	None	Cousin	None
1151. Samuel K., 16 yrs.; no vestige of hearing; vestibular responses diminished	Second cousins	None	One brother; one sister
1152. Celia K., 17 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	Two brothers
1153. Morris K., 15 yrs.; vestige of hearing; vestibular responses diminished			
1154. Sam F., 19 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister
1155. Oscar N., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1156. Leon A., 12 yrs.; vestige of hearing; vestibular responses normal	None	Father and mother	One sister
1157. Abraham E., 13 yrs.; no vestige of hearing; vestibular responses diminished	Cousins	Father's cousin; uncle	None
1158. Fannie F., 14 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	Three brothers
1160. Annie C., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1161. Mary L., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Paternal aunt; uncle	One brother
1162. George A., 14 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1163. Harry H., 16 yrs.; vestige of hearing; vestibular responses diminished	None	Mother	One brother; one sister
1164. Dorothy W., 9 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1165. John B., 7 yrs.; vestige of hearing; no vestibular response	Second cousins	None	None
1166. William C., 11 yrs.; vestige of hearing; no vestibular response	Second cousins	Father and mother	None
1167. Donald D., 7 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	Sister
1168. Sandy T., 15 yrs.; vestige of hearing; vestibular responses normal	Third cousins	Three uncles; one aunt	None
1169. Irving S., 16 yrs.; no vestige of hearing; vestibular responses diminished	None	Uncle	None
1170. Vladymir M., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1171. Gertrude D., 16 yrs.; vestige of hearing; no vestibular response	Second cousins	Aunt	One sister
1172. Tillie N., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1173. Dorothy, B., 18 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1174. Ida H., 17 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	None
1175. Philip B., 14 yrs.; no vestige of hearing; vestibular responses diminished	None	Mother	None
1176. Evelyn F., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
1177. Virginia D., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	One brother
1178. Sigmund W., 8 yrs.; no vestige of hearing; no vestibular response	Third cousins	None	None
1179. Leonard F., 12 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	Two brothers; one sister
1180. Ivan M., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers
1181. Andrew S., 15 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	None
1182. Mary C., 14 yrs.; vestige of hearing; no vestibular response	None	Father; mother; uncle; aunt; grandfather	One half-brother
1183. David Z., 14 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1184. Michael C., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1185. Louis B., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1186. Ethel K., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1187. Milton K., 18 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	One sister
1188. Irene G., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
1189. Myroslawa M., 13 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1190. Joseph N., 15 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
1191. Jacob N., 18 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1192. Angeline D., 19 yrs.; no vestige of hearing; vestibular responses diminished.	None	None	One brother; One sister
1193. Herman G., 7 yrs.; vestige of hearing; vestibular responses diminished	None	Cousins	Two brothers
1194. John L., 8 yrs.; no vestige of hearing; vestibular responses diminished	None	Greatuncle	None
1195. James C., 8 yrs.; vestige of hearing; no vestibular response	None	Father and mother	One brother; One sister
1196. Isaac N., 9 yrs.; vestige of hearing; vestibular responses normal	Cousins	None	None
1197. Esther V., 5 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	None
1198. Lillian L., 16 yrs.; vestige of hearing; vestibular responses normal	Cousins	None	None
1199. Rose R., 14 yrs.; no vestige of hearing; vestibular responses diminished	Second cousins	None	None
1200. Susie R., 13 yrs.; no vestige of hearing; no vestibular response	Second cousins	None	None
1201. Lillian P., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
1202. Geraldine Sh., 12 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother (in school); one sister
1203. Victor Sh., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	Two sisters
1204. Rose P., 6 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1205. Antoinette G., 7 yrs.; no vestige of hearing; vestibular response normal	None	None	One brother
1206. Lucy V., 7 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1207. Elsie K., 8 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1208. Salvatora L., 7 yrs.; no vestige of hearing; vestibular responses normal	None	One aunt	One sister
1209. Frances L., 11 yrs.; vestige of hearing; vestibular responses normal	None	One aunt	One sister
1210. Carmella A., 5 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1213. Santina P., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1214. Virginia Re., 7 yrs.; vestige of hearing; no vestibular response	None	None	Two brothers; one sister
1215. Rita Re., 12 yrs.; vestige of hearing; no vestibular response	None	None	Two brothers; one sister
1216. Antoinette Ma., 9 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1217. Ignatius Ma., 13 yrs.; no vestige of hearing; vestibular response normal	None	None	Two sisters
1218. Providence S., 8 yrs.; vestige of hearing; no vestibular response	None	Uncle	None
1219. Bertha D., 8 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1220. Helen P., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1221. Gertrude L., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1222. Germaine G., 11 yrs.; vestige of hearing; no vestibular response	Cousins	None	One brother
1223. Josephine M., 11 yrs.; vestige of hearing; vestibular responses normal	Cousins	None	None
1224. Anna B., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1225. Carl B., 14 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
1226. Lottie H., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1227. Charles H., 9 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister; both born deaf
1228. Benedict H., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister
1229. Margaret K., 12 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	None
1230. Eleanor T., 11 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	None

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1231. Stella W., 11 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	None
1232. Mary K., 19 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1233. Albertina C., 18 yrs.; no vestige of hearing; vestibular responses diminished	None	Second cousins	One brother
1234. John C., 13 yrs.; vestige of hearing; vestibular responses diminished	None	Second cousins	One sister
1235. Anna M., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers
1236. Ignatius M., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister
1237. Charles M., 20 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister
1238. Leona L., 19 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; three sisters
1239. Florence L., 18 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	Two sisters
1240. Angelina No., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers; two sisters
1241. Catherine Pi., 15 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother; one sister
1242. Vincent Pi., 17 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
1243. Margaret V., 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
1244. Margaret H., 16 yrs.; no vestige of hearing; no vestibular response	None	Cousin	None
1245. Teresa C., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1246. Dorothy A., 6 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1247. Rose Ca., 13 yrs.; no vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister
1248. Vincent Ca., 7 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister
1249. Rose Fa., 13 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	Two brothers
1250. John Fa., 11 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	One brother; one sister
1251. Nicholas Fa., 9 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One brother; one sister

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1252. Ulda Fi., 12 yrs.; vestige of hearing; no vestibular responses	None	None	One brother; one sister
1253. Helen M., 11 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One brother
1254. Peter Ca., ? yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1255. Liborio Ca., 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1256. Anthony D., 18 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1257. John Ca., 14 yrs.; vestige of hearing; no vestibular response	None	None	Two brothers
1258. Bernard Lo., 17 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One sister
1259. Lluster Co., 16 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	None
1260. Michael L., 16 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	One brother
1261. Joseph La., 12 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One brother
1262. William Br., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1263. Thomas B., 18 yrs.; no vestige of hearing; vestibular responses normal	None	None	Aunt
1264. Frederick Or., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers; one sister
1265. Benedicto Mo., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister
1266. Vito Mo., 8 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister; both born deaf
1267. John Do., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None
1268. Julius Va., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two brothers
1269. Joseph Va., 12 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	One brother
1270. Paul La., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1271. Vincent Ma., 15 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother
1272. Amadeo Ma., 11 yrs.; no vestige of hearing; vestibular responses normal	None	None	One brother

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1273. Angelo Ce., 14 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
1274. Frank Sc., 15 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One sister
1275. Walter Wo., ? yrs.; vestige of hearing; vestibular responses normal	None	None	One brother
1276. John Di., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1277. Vincent De., 10 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1278. Victor Sh., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	Two sisters
1279. Ralph Re., 11 yrs.; vestige of hearing; no vestibular response	None	None	One brother; two sisters
1280. Tobias Di., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1281. Joseph La., 11 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
1282. Romeo Gr., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1283. Nicholas Sa., 10 yrs.; no vestige of hearing; no vestibular response	None	None	Two brothers
1284. Raphael Sa., 8 yrs.; vestige of hearing; no vestibular response	None	None	Two brothers
1285. Peter Dm., 11 yrs.; vestige of hearing; vestibular response normal	First cousins	None	None
1286. Daniel Co., 9 yrs.; vestige of hearing; vestibular responses normal	Second cousins	None	None
1287. Michael Pl., 9 yrs.; vestige of hearing; vestibular response normal	None	None	One brother; one sister; both born deaf
1288. Benedetto Pa., 8 yrs.; vestige of hearing; no vestibular response	First cousins	None	One sister
1289. Henry De., 8 yrs.; no vestige of hearing; no vestibular response	First cousins	None	One sister (born deaf)
1290. Frederich De., 7 yrs.; no vestige of hearing; no vestibular response	None	Two cousins (born deaf)	None
1291. Michael Bu., 9 yrs.; vestige of hearing	None	Three uncles	None
1292. Anthony L., 7 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	None
1293. Pasquaele Ru., 8 yrs.; no vestige of hearing; vestibular responses normal	First cousins	None	One brother; one sister (born deaf)

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1294. Francis Ke., 4 yrs.; no vestige of hearing; vestibular responses normal	None	None	One sister
1295. Alphonse Do., 4 yrs.; vestige of hearing; vestibular responses normal	None	Father; mother; other relatives	None
1296. Felix N., 15 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1297. Ruth L., 12 yrs.; vestige of hearing; vestibular responses weak	None	Paternal uncle (cause unknown)	One brother; one sister
1298. William D., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1299. Mary D., 10 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1300. George N., 7 yrs.; no vestige of hearing; no vestibular response	None	Maternal grandfather, born deaf	None
1301. Rose B., 6 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister (born deaf)
1302. Betty M., 7 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1303. Frank M., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1304. Doris R., 7 yrs.; vestige of hearing; vestibular responses diminished	None	Father and mother	None
1305. Saul S., 8 yrs.; no vestige of hearing; no vestibular response	None	Maternal second cousins	None
1306. Elliott S., 7 yrs.; no vestige of hearing; no vestibular response	None	Maternal grandfather; second cousin	One sister
1307. Shirley S., 11 yrs.; no vestige of hearing; no vestibular response	None	Maternal grandfather; second cousin	One brother
1308. Leo J., 8 yrs.; vestige of hearing; vestibular responses diminished	None	Three second cousins (born deaf)	None
1309. Ellen S., 7 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1310. Phillis S., 16 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1311. Bertha B., 8 yrs.; no vestige of hearing; no vestibular response	Cousins	None	Two sisters (born deaf)
1312. Bella B., 10 yrs.; no vestige of hearing; no vestibular response	Cousins	None	Two sisters (born deaf)
1313. Bessie B., 14 yrs.; vestige of hearing; no vestibular response	Cousins	None	Two sisters (born deaf)
1314. George G., 8 yrs.; vestige of hearing; no vestibular response	Cousins	None	One sister (born deaf)
1315. Meyer P., 8 yrs.; no vestige of hearing; vestibular responses diminished	None	Maternal uncle (born deaf); two cousins (born deaf)	One sister (born deaf)
1316. Harry P., 17 yrs.; vestige of hearing; vestibular responses normal	None	Maternal uncle (born deaf); two cousins (born deaf)	One sister (born deaf)

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1317. Malvene F., 8 yrs.; no vestige of hearing; vestibular responses diminished	None	Father and mother	None
1318. Louis F., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters (born deaf)
1319. Norma F., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother, one sister (both born deaf)
1320. Anna N., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1321. Abraham G., 10 yrs.; vestige of hearing; vestibular responses diminished	Second cousins	None	None
1322. Gaintano S., 8 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1323. Katherine G., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Paternal second cousins (born deaf)	None
1324. Sam K., 12 yrs.; vestige of hearing; vestibular responses normal	None	Paternal uncle (cause unknown)	None
1325. Ruth S., 11 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1326. Rose S., 11 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1327. Carlo D., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister (born deaf)
1328. Catherine D., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother (born deaf)
1329. Sidney M., 14 yrs.; vestige of hearing; no vestibular response	First cousins	None	None
1330. Henry C., 14 yrs.; no vestige of hearing; no vestibular response	None	Two maternal great-uncles	None
1331. Marvin K., 11 yrs.; no vestige of hearing; vestibular responses diminished	Distantly related	None	One brother (cause unknown)
1332. Mamie Gl., 13 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother (born deaf)
1333. Slyvia Ml., 13 yrs.; vestige of hearing; vestibular responses normal	None	Maternal second cousins are deaf mutes	None
1334. Alfred So., 12 yrs.; vestige of hearing; vestibular responses diminished	None	Father; mother; maternal uncle	Two sisters (born deaf)
1335. Lillie So., 14 yrs.; vestige of hearing; vestibular responses diminished	None	Father; mother; maternal uncle	One brother; one sister (both born deaf)
1336. Ruth Ma., 17 yrs.; vestige of hearing; no vestibular response	Second cousins	None	One sister (born deaf)
1337. Helen Ma., 4 yrs.; no vestige of hearing; no vestibular response	Second cousins	None	One sister (born deaf)

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1338. Benjamin Mo., 17 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister (both born deaf)
1339. Fanny Pa., 17 yrs.; vestige of hearing; vestibular responses diminished	First cousins	None	One brother; one sister (both born deaf)
1340. Lillian Pa., 16 yrs.; vestige of hearing; no vestibular response	First cousins	None	One brother; one sister (both born deaf)
1341. Anna Ki., 11 yrs.; vestige of hearing; vestibular responses normal	None	None	Two brothers; one sister (all born deaf)
1342. Hyman Ki., 14 yrs.; no vestige of hearing; no vestibular response	None	None	Two sisters; one brother (all born deaf)
1343. Benjamin Fi., 12 yrs.; vestige of hearing; vestibular responses diminished	None	None	None
1344. Clara Co., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One sister
1345. Julius Go., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (cause unknown)
1346. Florence Br., 14 yrs.; vestige of hearing; vestibular response normal	None	None	One sister (born deaf)
1347. William Ep., 15 yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1348. Gertrude Ka., 14 yrs.; vestige of hearing; no vestibular response	None	None	One brother (born deaf)
1349. Sbarra Do., 14 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother (born deaf)
1350. Anna We., 13 yrs.; vestige of hearing; vestibular responses normal	First cousins	None	None
1351. Rose De St., 15 yrs.; vestige of hearing; vestibular responses normal	None	None	One brother; one sister (both born deaf)
1352. Ludwig Kr., 17 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	None
1353. Rebecca Be., 16 yrs.; no vestige of hearing; vestibular responses diminished	Cousins	Paternal uncle (cause unknown)	One brother (born deaf)
1354. Frank Po., 18 yrs.; no vestige of hearing; vestibular responses diminished	First cousins	None	None
1355. Abe Go., 19 yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister (born deaf)
1356. Helen Od., 17 yrs.; no vestige of hearing; vestibular responses diminished	Distant cousins	None	One sister (hard of hearing)

TABLE 6.—*Relation of Heredity and Consanguinity to Deafness in One Thousand Three Hundred and Seventy-Four Children—Continued*

Observations in Individual Cases

Cases	Nature of Consanguinity	History of Deafness in Family of Parents	Other Cases of Deafness in Immediate Family
1357. Ray Co., 17 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother (born deaf)
1358. Ralph Ka., 19 yrs.; vestige of hearing; vestibular responses diminished	Cousins	None	None
1359. Ira Le., 6 yrs.; vestige of hearing; vestibular responses diminished	None	Second cousins on both sides	None
1360. Marion Fi., 9 yrs.; vestige of hearing; vestibular responses diminished	None	None	Two sisters
1361. Helen Sc., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother; one sister
1362. Robert No., 10 yrs.; vestige of hearing; vestibular responses diminished	None	None	One brother
1363. Martha Da., 10 yrs.; vestige of hearing; vestibular responses diminished	None	Mother's cousin	None
1364. Mary Ci., ? yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother
1365. Helen Br., ? yrs.; vestige of hearing; vestibular responses diminished	None	None	One sister
1366. William To., ? yrs.; no vestige of hearing; vestibular responses diminished	None	Maternal cousin	None
1367. George Bi., ? yrs.; no vestige of hearing; vestibular responses diminished	None	None	One brother

This table needs no particular comment. People who have congenital deafness in the family should not bear offspring. The marriage of a normal person with one from a family suffering from congenital deafness does not, of course, carry the same degree of risk as does the intermarriage between two such families. Acquired deafness usually means some accident or infection. Such persons will not, of course, transmit their affliction to offspring. It is only in the congenitally deaf that this menace exists. The same does not hold true for the cases in which severe deafness is acquired in adult life, for here one is confronted with a disease (otosclerosis) in which heredity plays a conspicuous factor, but in which the defect in hearing does not begin to show, as a rule, until adult life.

The status of the vestibular responses in these children, in whom heredity and consanguinity are factors, is of particular interest, because it has usually been assumed that in congenital deafness the develop-

mental anomaly responsible for this condition involves only the cochlea, leaving the vestibular mechanism intact.

In this group of children, 445 had normal vestibular responses, 668 showed diminished responses, while in 252 no vestibular responses could be elicited. In 5 children it was not feasible to make the vestibular tests.

TABLE 7.—*Observations in Three Thousand, Three Hundred and Thirty-Four Cases (62.34 Per Cent) of Congenital Deafness*

- Definitely congenital 2,623 (78.67%)
  - Total deafness 1,018 (38.84%)
    - Other cases in family 430 (43.22%)
      - Vestibular reactions
        - Normal .. 154 (35.81%)
        - Diminished 184 (42.79%)
        - Absent ... 92 (21.40%)
    - No other cases in family 588 (56.78%)
      - Vestibular reactions
        - Normal .. 142 (24.15%)
        - Diminished 243 (41.32%)
        - Absent ... 199 (33.84%)
        - No record. 4 (0.69%)
  - Partial deafness 1,605 (61.16%)
    - Other cases in family 665 (41.45%)
      - Vestibular reactions
        - Normal .. 245 (36.84%)
        - Diminished 335 (50.39%)
        - Absent ... 84 (12.63%)
        - No record. 1 (0.14%)
    - No other cases in family 940 (58.55%)
      - Vestibular reactions
        - Normal ... 350 (37.27%)
        - Diminished 432 (46.00%)
        - Absent ... 151 (16.08%)
        - No record. 7 (0.65%)
- No record, 4 1.19%
- Probably congenital 707 (20.14%)
  - Total deafness 243 (34.37%)
    - Other cases in family 47 (19.34%)
      - Vestibular reactions
        - Normal .. 12 (25.53%)
        - Diminished 21 (44.68%)
        - Absent ... 14 (29.79%)
    - No other cases in family 196 (80.66%)
      - Vestibular reactions
        - Normal .. 36 (18.36%)
        - Diminished 94 (47.96%)
        - Absent ... 65 (33.17%)
        - Not tested. 1 (0.51%)
  - Partial deafness 464 (65.63%)
    - Other cases in family 83 (17.88%)
      - Vestibular reactions
        - Normal .. 32 (38.55%)
        - Diminished 41 (49.38%)
        - Absent ... 10 (12.07%)
    - No other cases in family 381 (82.12%)
      - Vestibular reactions
        - Normal .. 124 (32.54%)
        - Diminished 192 (50.40%)
        - Absent ... 65 (17.06%)

CONGENITAL DEAFNESS

In table 7 we have undertaken to analyze the statistics in the 3,334 children classed as congenitally deaf. For reasons that have already been stated, data regarding the congenital origin of deafness are particularly liable to be misleading. For this reason we have kept special

data regarding children in whom we felt sure of the congenital nature of the deafness, and we have compiled another table for those in whom we felt some doubt of the congenital origin. Deafness was definitely congenital in 2,623 children and probably congenital in 707. I shall

TABLE 8.—*Observations in Two Thousand and Fourteen Cases (37.66 Per Cent) of Acquired Deafness*

Associated at onset with otitis media 426 (21.14%)	Definitely acquired 335 (78.63%)	Total deafness 100 (29.85%)	Vestibular reactions: Normal ... 16 (16.00 %); Diminished. 29 (29.00 %); Absent .... 55 (55.00 %)
		Partial deafness 235 (70.15%)	Vestibular reactions: Normal ... 100 (42.55 %); Diminished. 95 (40.42 %); Absent .... 38 (16.17 %); Not tested. 2 (0.86 %)
	Probably acquired 91 (21.27%)	Total deafness 37 (40.66%)	Vestibular reactions: Normal ... 14 (37.83 %); Diminished. 12 (32.43 %); Absent .... 11 (29.74 %)
		Partial deafness 54 (59.34%)	Vestibular reactions: Normal ... 21 (38.887%); Diminished. 23 (42.591%); Absent 10 (18.518%)
1 record of no value 0.07%			
Not associated at onset with otitis media 1,587 (78.79%)	Definitely acquired 1,215 (76.55%)	Total deafness 524 (43.12%)	Vestibular reactions: Not tested. 1 (0.19 %); Normal ... 54 (10.30 %); Diminished. 146 (27.76 %); Absent .... 322 (63.35 %)
		Partial deafness 691 (56.88%)	Vestibular reactions: Normal ... 163 (23.59 %); Diminished. 306 (44.14 %); Absent .... 220 (31.84 %); Not tested. 1 (0.45 %)
	Probably acquired 372 (23.45%)	Total deafness 160 (43.01%)	Vestibular reactions: Normal ... 16 (10.00 %); Diminished. 68 (42.41 %); Absent .... 76 (47.59 %)
		Partial deafness 212 (56.99%)	Vestibular reactions: Normal ... 55 (25.94 %); Diminished. 108 (50.90 %); Absent .... 48 (22.58 %); Not tested. 1 (0.58 %)

discuss here only the first group. Of these 1,018, or 38.84 per cent, were totally deaf and 1,605, or 61.16 per cent, partially deaf. Of the children who were found to be totally deaf, 430 belonged to families in which there were other cases of deafness, while 588 did not. Of children showing partial deafness, 665 belonged to families in which there were other cases of deafness, while 940 did not. For reasons that have been stated, the status of the vestibular mechanism in this

group of congenitally deaf children is of particular interest. Of the 430 totally deaf children in whose families there were cases of deafness, 154 gave normal vestibular responses, 184 gave diminished responses and only 92 failed to give these responses. Of the 588 children with total deafness but in whose families there were no other cases, 142 gave normal responses, 243 diminished responses and 199 no responses. For 4 children, no vestibular tests were recorded. Of the 665 who had partial congenital deafness and in whose families there were other cases of deafness, vestibular responses were normal in 245, diminished in 335 and completely absent in 84; for 1 child no record was made. Of the 940 partially congenitally deaf children, no other members of whose families were deaf, vestibular responses were found to be normal in 350, diminished in 432 and absent in 151; in 7 cases the responses were not recorded.

ACQUIRED DEAFNESS

We found 2,014 cases of acquired deafness—37.66 per cent of the entire number; 426 of these, or 21.14 per cent, were associated with otitis media at the onset of the deafness and 1,587, or 78.79 per cent, developed without concurrent otitis media. I would point out again that we are not warranted in the conclusion that an associated otitis media necessarily had anything to do with the occurrence of the internal ear disease which alone is capable of producing deafness severe enough to necessitate the child's being placed in an institution for the deaf. In some of these children the involvement of the labyrinth, no doubt, was in the nature of a labyrinthitis set up through an extension of the suppurative otitis media into the internal ear. We divided both of these groups into cases in which we were sure the deafness had been acquired and cases in which we felt that it had probably been acquired but in which there was an element of doubt.

There were 100 totally deaf children in the group of 335 who had definitely acquired deafness associated at onset with otitis media. Vestibular responses were normal in 16 of these children, diminished in 29 and absent in 55. There were 235 partially deaf children in this group with definitely acquired deafness which was associated at onset with otitis media. Vestibular responses were normal in 100, diminished in 95, absent in 38 and not tested in 2. These figures may be compared with the statistics on the 1,215 children with definitely acquired deafness which was not associated at onset with otitis media; 524 of these were totally deaf, and the vestibular responses in this group were normal in 54, diminished in 146, absent in 322 and not tested in 1; 691 in this group were partially deaf, and gave the following vestibular reactions: normal in 163, diminished in 306, absent in 220 and not tested in 1.

STATUS OF THE HEARING

Table 9 shows the status of hearing in the 5,348 children examined; 2,083, or 38.91 per cent, were found to be totally deaf, and 3,260, or 60.95 per cent, partially deaf. In 5 the records of hearing were of no value. Of the 2,083 totally deaf children, 1,262 had congenital and

TABLE 9.—*States of Hearing in Five Thousand, Three Hundred and Forty-Eight Cases*

Total deafness 2,083 (38.91%)	Congenital deafness, 1,262 (60.58%)		Vestibular reactions: Normal ... 344 (27.46 %); Diminished. 542 (42.94 %); Absent .... 370 (29.31 %); Not tested. 6 (0.29 %)
	Acquired deafness 821 (39.42%)	Associated with otitis media 137 (16.69%)	Normal ... 30 (21.9 %); Vestibular reactions: Diminished. 41 (30.0 %); Absent .... 66 (48.1 %)
		Not associated with otitis media 684 (83.31%)	Vestibular reactions: Normal ... 70 (10.23 %); Diminished. 214 (31.28 %); Absent .... 398 (58.18 %); Not tested. 2 (0.31 %)
5 records of no value 0.14%			
Partial deafness 3,260 (60.95%)	Congenital deafness, 2,068 (63.43%)		Vestibular reactions: Normal ... 651 (31.43 %); Diminished. 1,000 (48.35 %); Absent .... 310 (19.882%); Not tested.. 7 (0.338%)
	Acquired deafness 1,192 (36.57%)	Associated with otitis media 289 (24.24%)	Vestibular reactions: Normal ... 121 (41.98 %); Diminished. 118 (40.83 %); Absent .... 48 (16.60 %); Not tested.. 2 (0.49 %)
		Not associated with otitis media 903 (75.76%)	Vestibular reactions: Normal ... 218 (24.14 %); Diminished. 411 (45.51 %); Absent .... 270 (29.90 %); Not tested.. 4 (0.45 %)

821 acquired deafness. Of the 3,260 children who were partially deaf 2,068 had congenital and 1,192 acquired deafness. This table gives an analysis of the status of the vestibular reactions in each group.

SCHWABACH TEST

The status of bone conduction is known to be an important matter in cases of deafness, particularly because of the relation between bone conduction and the voice. A person suffering from defective hearing in whom this defect is the result of obstruction in sound conduction has a bone conduction prolonged above the normal. There is a peculiar

TABLE 10.—*Results of the Schwabach Test*

- Bone conduction absent 3,249 (60.75%)
  - Total defect with audiometer 778 (23.95%)
    - Congenital 440 (56.55%)
    - Acquired 338 (43.45%)
      - Associated with otitis media 55 (17.58%)
      - Not associated with otitis media 283 (83.42%)
  - No record, 9 (0.27%)
  - Remnant of hearing with audiometer 2,462 (75.78%)
    - Congenital 1,621 (65.84%)
    - Acquired 841 (34.16%)
      - Associated with otitis media 151 (17.96%)
      - Not associated with otitis media 690 (82.04%)
- Bone conduction shortened 1,460 (27.48%)
  - Total defect with audiometer 100 (6.50%)
    - Congenital 66 (66%)
    - Acquired 34 (34%)
      - Associated with otitis media 6 (21.42%)
      - Not associated with otitis media 28 (78.38%)
  - No record, 3 (0.5%)
  - Remnant of hearing with audiometer 1,357 (92.94%)
    - Congenital 813 (59.91%)
    - Acquired 534 (40.09%)
      - Associated with otitis media 125 (25.29%)
      - Not associated with otitis media 409 (74.71%)
- No record, 148 (2.78%)
- Bone conduction normal 410 (7.66%)
  - Total defect with audiometer 8 (1.95%)
    - Congenital 4 (50%)
    - Acquired 4 (50%)
      - Associated with otitis media 0
      - Not associated with otitis media 4 (100%)
  - Remnant of hearing with audiometer 402 (98.05%)
    - Congenital 225 (55.97%)
    - Acquired 177 (44.03%)
      - Associated with otitis media 57 (32.20%)
      - Not associated with otitis media 120 (67.80%)
- Bone conduction prolonged 81 (1.33%)
  - Total defect with audiometer 3 (3.71%)
    - Congenital 1 (33.34%)
    - Acquired 2 (66.66%)
      - Associated with otitis media 0
      - Not associated with otitis media 2 (100%)
  - Remnant of hearing with audiometer 78 (96.29%)
    - Congenital 37 (47.43%)
    - Acquired 41 (52.87%)
      - Associated with otitis media 13 (43.9%)
      - Not associated with otitis media 18 (56.1%)

alteration in the voice of these persons. There is a tendency to speak too low, because the increased bone conduction makes them hear their own voices better than can a normal hearing person. On the other hand, a person suffering from defective hearing as a result of changes in the nervous mechanism has shorter bone conduction than normal, and in these people there is no tendency to hear their own voice too loudly; when the deafness is marked, the voice is prone to take on a harsh metallic sound, of which the patient is not aware. The Schwabach test was carried out in this work by using a Bezold-Edelmann fork which was allowed to rest with its own weight on the calvarium. In the 5,348 children examined, bone conduction was absent in 3,249, or 60.75 per cent, shortened in 1,460, or 27.48 per cent, and normal in 410, or 7.66 per cent. It was actually prolonged beyond the normal in 81 children, and in 148 the response seemed so doubtful as to have no value.

Table 10 presents an analysis of the statistics of the Schwabach test. Of the 3,249 children showing absence of bone conduction, 778 showed total defect in hearing when using the audiometer, 2,462 showed remnants of hearing, and in 9 the observations were not accurate enough to be recorded. Of the 1,460 children with shortened bone conduction, 100 showed total deafness in audiometer tests, and 1,357 showed remnants of hearing. Of the 410 children with normal bone conduction, 8 showed total deafness in the audiometer tests and 402 showed remnants of hearing. Of the 81 children in whom bone conduction was actually prolonged, 3 gave no response to the audiometer tests, while 78 showed remnants of hearing.

I shall not attempt to explain all of these observations, but I would point out that it is not uncommon in cases in which there is profound deafness with extensive involvement of the nervous mechanism, as in the advanced stages of otosclerosis, to find the existence of a real prolongation of bone conduction resulting from stapes fixation. Of the 81 cases that showed prolonged bone conduction with remnants of hearing for the audiometer, 37 were congenital and 41 acquired. Only 19 of the latter were associated with an otitis media that might be held responsible for an associated obstruction in sound conduction sufficient to account for a prolonged bone conduction. I have no suggestion to offer in explanation of the prolonged bone conduction in the 37 cases of congenital deafness in this group.

RINNE TEST

As was stated in our previous reports, the Rinne test is of such paramount importance for the otologist in differentiating clinically between deafness due to obstruction in sound conduction and deafness due to a degeneration of the nervous mechanism, that we have under-

taken to compile some statistics regarding this test in the children we have examined. These statistics are graphically shown in table 11. Not all children could be tested satisfactorily in this way, and the figures given represent the number of ears tested rather than the number of

TABLE 11.—*Results of the Rinne Test*

- **Positive Rinne reaction 966 (16.95%)**
  - **Unilateral 339 (35.09%)**
    - Total defect with audiometer, 23 (6.8%)
    - Remnant of hearing with audiometer 315 (92.91%)
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 121 (38.41%)
      - Above and below 2,000 double vibrations 194 (61.59%)
    - No record, 1 (0.29%)
  - **Bilateral 627 (64.91%)**
    - Total defect with audiometer, 16 (5.58%)
    - Remnant of hearing with audiometer 591 (94.42%)
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 152 (25.72%)
      - Above and below 2,000 double vibrations 439 (74.28%)
- **Negative Rinne reaction 662 (11.63%)**
  - **Unilateral 232 (35.04%)**
    - Total defect with audiometer, 9 (3.92%)
    - Remnant of hearing with audiometer 223 (96.08%)
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 141 (63.22%)
      - Above and below 2,000 double vibrations 82 (36.78%)
  - **Bilateral 430 (64.96%)**
    - Total defect with audiometer, 5 (1.16%)
    - Remnant of hearing with audiometer 425 (98.84%)
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 161 (38.37%)
      - Above and below 2,000 double vibrations 263 (61.88%) 1 not tested (0.05%)
- **Absolute negative Rinne reaction 4,069 (71.42%)**
  - **Unilateral 515 (12.41%)**
    - Total defect with audiometer, 66 (12.82%)
    - Remnant of hearing with audiometer 449 (87.18%)
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 254 (56.57%)
      - Above and below 2,000 double vibrations 195 (43.43%)
  - **Bilateral 3,554 (87.59%)**
    - Total defect with audiometer, 778 (21.90%)
    - Remnant of hearing with audiometer 2,773 (78.85%)
      - Above 2,000 double vibrations 1 (0.04%)
      - Below 2,000 double vibrations 2,011 (72.52%)
      - Above and below 2,000 double vibrations 761 (27.44%)
    - No record, 3 (0.05%)

children examined. For example, a positive Rinne reaction was obtained in 966 instances, a negative reaction in 662 and an absolutely negative result—that is, the fork was heard only by bone conduction—in 4,069. In this table, for obvious reasons, each group of cases is divided into those in which the reaction was unilateral and those in which it was

bilateral. The status of the hearing when tests were made with the audiometer is also recorded: first, whether there was any hearing, and, second, when hearing was found by the audiometer, whether this remnant was for tones above or below 2,000 double vibrations or whether it was for tones both above and below this tone. It is interesting to note that only in a single instance was a remnant of hearing found

TABLE 12.—*Relation of Audiometer Reactions to Other Tests*

- Total deafness for all tests 736 (13.99%)
  - Marked speech defect, 556 (75.55%)
  - Without marked speech defect, 180 (24.45%)
- Remnants of hearing 4,351 (82.57%)
  - With audiometer and other tests 3,013 (67.2%)
    - Marked speech defect 1,543 (51.21%)
      - Remnants:
        - Above 2,000 double vibrations 1 (0.06%)
        - Below 2,000 double vibrations 793 (51.38%)
        - Above and below 2,000 double vibrations 749 (48.56%)
    - Without marked speech defect 1,470 (48.79%)
      - Remnants:
        - Above 2,000 double vibrations
        - Below 2,000 double vibrations 523 (35.58%)
        - Above and below 2,000 double vibrations 947 (64.42%)
  - With audiometer; other tests negative 1,306 (30.0%)
    - Marked speech defect 939 (71.9%)
      - Remnants:
        - Above 2,000 double vibrations 1 (0.1%)
        - Below 2,000 double vibrations 840 (89.45%)
        - Above and below 2,000 double vibrations 97 (10.45%)
      - Not tested, 1
    - Without marked speech defect 367 (28.1%)
      - Remnants:
        - Above 2,000 double vibrations
        - Below 2,000 double vibrations 315 (85.83%)
        - Above and below 2,000 double vibrations 52 (14.17%)
  - Hearing in other tests; audiometer negative, 122 (2.8%)

No audiograms, 23 (0.43%)
No record of speech, 148 (3.01%)

only for tones above 2,000 double vibrations. In all other cases, the hearing was found to be either for tones below this tone or both above and below it.

THE RELATION BETWEEN AUDIOMETER REACTIONS AND OTHER HEARING TESTS

Statistics for table 12 were compiled from the examination of 5,087 children; 736 of these showed total deafness in all tests, that is, with audiometer, voice, tuning forks, and whistles by air conduction; 556

showed a marked speech defect whereas the speech defect in 180 was not marked. Remnants of hearing were found in 4,351; in 3,013 of these the remnants of hearing were detected by the audiometer as well as by other tests; in 1,306 the audiometer showed remnants of hearing which were not found by other tests, and 122 showed remnants of hearing in other tests but gave negative results with the audiometer.

Of 3,812 children totally deaf to the voice, 845, or 22.16 per cent, were also totally deaf in the audiometer tests; 2,944, or 77.24 per cent, showed remnants of hearing in the audiometer test. The table

TABLE 13.—*Total Deafness for Voice in Three Thousand, Eight Hundred and Twelve Children (71.28 Per Cent)*

Total deafness with audiometer 845 (22.16%)	Total deafness with forks; bone and air conduction, 725 (85.69%)		Congenital....	415 (57.24%)
			Acquired.....	310 (42.76%)
	Remnants of hearing with forks 120 (14.31%)	Bone conduction 19 (15.83%)	Congenital....	11 (57.89%)
			Acquired.....	8 (42.11%)
		Air conduction 101 (84.17%)	Congenital....	73 (72.27%)
			Acquired.....	28 (27.73%)
No record, 23 (0.6%)				
Remnants of hearing with audiometer 2,944 (77.24%)	Total deafness with forks; bone and air conduction, 1,345 (45.69%)		Congenital....	830 (61.71%)
			Acquired.....	515 (38.29%)
	Remnants of hearing with forks 260 (20.4%) 1,599 (54.31%)	Bone conduction 442 (27.64%)	Congenital....	211 (47.73%)
			Acquired.....	231 (52.27%)
		Air conduction 1,157 (72.36%)	Congenital....	761 (65.77%)
			Acquired.....	396 (34.23%)

shows further the status of hearing for both bone and air conduction and whether the deafness was congenital or acquired.

Table 14 shows an analysis of 2,083 children who had no marked speech defect; 180 of these were totally deaf in all tests, 1,470 showed remnants of hearing in the audiometer as well as other tests, 367 were totally deaf in other tests but showed remnants of hearing in the audiometer test, and in 53 there was total deafness in the audiometer test but remnants of hearing in other tests. For 13 children no audiograms were obtained. The status of bone conduction in these statistics is of particular interest. Of the 1,470 children with remnants of hearing in the audiometer and other tests, bone conduction was present in 1,063 and absent in 407. Of the 53 children showing total deafness in the audiometer test, but with remnants of hearing in other tests, bone conduction was present in 36 and absent in 17.

TABLE 14.—*Two Thousand Eighty-Three Children (38.94 Per Cent) Without Marked Speech Defect*

Total deafness to all tests, 180 (8.74%)

- Remnants with audiometer and other tests 1,470 (70.49%)
  - Bone conduction present 1,063 (72.32%)
    - Hearing remnant:
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 310 (29.16%)
      - Above and below 2,000 double vibrations 753 (70.84%)
  - Bone conduction absent 407 (27.68%)
    - Hearing remnant:
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 213 (52.33%)
      - Above and below 2,000 double vibrations 194 (47.67%)
- Total deafness in other tests; remnants in audiometer test, 367 (17.61%)
  - Hearing remnant:
    - Above 2,000 double vibrations
    - Below 2,000 double vibrations 315 (85.83%)
    - Above and below 2,000 double vibrations 52 (14.17%)
- Total deafness with audiometer; remnant in other tests 53 (2.54%)
  - Bone conduction present 36 (67.92%)
  - Bone conduction absent 17 (32.08%)

No audiograms, 13 (0.62%)

TABLE 15.—*Marked Speech Defect in Three Thousand, One Hundred and Twenty-Five Children (61.06 Per Cent)*

Total deafness in all tests, 556 (17.78%)

- Remnants with audiometer and other tests 1,543 (44.24%)
  - Bone conduction present 801 (51.91%)
    - Hearing remnant:
      - Above 2,000 double vibrations 1 (0.12%)
      - Below 2,000 double vibrations 424 (52.93%)
      - Above and below 2,000 double vibrations 376 (46.95%)
  - Bone conduction absent 742 (48.09%)
    - Hearing remnant:
      - Above 2,000 double vibrations
      - Below 2,000 double vibrations 369 (49.72%)
      - Above and below 2,000 double vibrations 373 (50.28%)
- Total deafness in other tests; remnants in audiometer test, 939 (30.72%)
  - Hearing remnant:
    - Above 2,000 double vibrations 1 (0.10%)
    - Below 2,000 double vibrations 840 (89.47%)
    - Above and below 2,000 double vibrations 97 (10.33%)
    - Not tested, 1 (0.10%)
- Total deafness in audiometer tests; remnants in other tests 69 (2.2%)
  - Bone conduction present 33 (47.82%)
  - Bone conduction absent 36 (52.18%)

No record of speech, 140 (4.48%)
No audiograms, 18 (0.58%)

Table 15 gives the statistics on 3,125 children in whom there was a marked speech defect. Speech defects in some form or degree are present in most of the children in these institutions. We use the term "marked speech defect" only for those persons who mouth their words in such a manner that the untrained listener experiences considerable difficulty in understanding them. The presumption would be that a more or less definite relation exists between the degree of speech defect and the degree of deafness, and especially between the presence or absence of bone conduction and such defects. Of particular interest in this connection is the question of the relation between the existence of remnants of hearing as detected by the audiometer for those parts of the tone scale most necessary for the hearing of the human voice. Of the 3,125 children in this group, 556 were totally deaf for all tests. Remnants of hearing in the audiometer and other tests were found in 1,543 children. Total deafness in other tests but remnants of hearing in the audiometer test were found in 939 children. Total deafness in the audiometer test with remnants of hearing in other tests was found in 69 children. Of the 1,543 children with remnants of hearing in both the audiometer and other tests, bone conduction was present in 801 and absent in 742. Of the 69 with remnants of hearing in other tests but none in the audiometer test, bone conduction was present in 33 and absent in 36. In only 2 of these cases was the remnant of hearing in the audiometer test restricted to the region above 2,000 vibrations. All the other cases were about equally divided between those in which the remnant was found to be only for tones above or both above and below 2,000 double vibrations. An exception was found in the children with total deafness in other tests but with remnants of hearing in the audiometer test; of the 939 children in this group, 840 showed remnants of hearing confined to the region below 2,000 double vibrations and only 97 showed remnants both above and below 2,000 double vibrations.

VESTIBULAR RESPONSES TO TURNING IN THEIR REACTION TO DEAFNESS

In table 16 we have undertaken an analysis of the vestibular responses to turning in 5,348 children, (1) in relation to congenital and acquired deafness and (2) in relation to total or partial deafness. Vestibular responses to turning were obtained in 3,870 children, or 72.36 per cent. Responses were absent in 1,461, or 25.64 per cent. In 15 children the tests were of no value and in 92 no tests were undertaken. Of the 3,870 patients showing vestibular respo...es, 2,559, or 66.09 per cent, had congenital deafness, while 1,221, or 33.91 per cent, had acquired deafness. In the congenital group of 2,559 giving vestibular responses, 857, or 33.50 per cent, were totally deaf; 1,701, or 66.47 per

cent, were partially deaf. Of the 1,461 children giving no vestibular response to turning, 676 had congenital deafness and 785 acquired deafness. Of the 676 congenitally deaf children, the deafness was total in 356 and partial in 320. Without attempting to analyze all the data brought out in the table, I call attention to the fact that vestibular responses were found about twice as frequently in congenitally deaf children as in those with acquired deafness. In both groups of cases, that is, of congenital and of acquired deafness, vestibular responses

TABLE 16.—*Status of Vestibular Mechanism in Five Thousand, Three Hundred and Forty-Eight Cases*

Vestibular response present 3,780 (72.36%)	Congenital deafness, 2,559 (66.09%)		Total deafness	857 (33.50%)
			Partial deafness	1701 (66.47%)
			Not tested	1 (0.03%)
	Acquired deafness 1,221 (33.91%)	Associated with otitis media 335 (27.44%)	Total deafness	76 (22.98%)
			Partial deafness	259 (77.02%)
		Not associated with otitis media 886 (72.56%)	Total deafness	277 (31.28%)
			Partial deafness	609 (68.72%)
15 (0.28%) records of no value				
92 (1.72%) not tested				
Vestibular response absent 1,461 (25.64%)	Congenital deafness, 676 (45.59%)		Total deafness	356 (52.66%)
			Partial deafness	320 (47.34%)
	Acquired deafness 785 (54.41%)	Associated with otitis media 110 (15.29%)	Total deafness	70 (63.68%)
			Partial deafness	60 (36.37%)
		Not associated with otitis media 655 (84.71%)	Total deafness	399 (60.91%)
			Partial deafness	255 (38.93%)
			Not tested	1 (0.14%)

were present in about twice as many children with partial deafness as in children with total deafness. The outstanding facts are that vestibular responses were found in both congenital and acquired deafness and in both total and partial deafness. Likewise, vestibular responses were absent in both congenital and acquired deafness and in very nearly the same proportion, slightly more in the acquired cases. Vestibular responses were absent in both the congenital and the acquired groups in about the same proportion whether the deafness was total or partial.

CALORIC REACTIONS

Table 17 shows the relation of the caloric reactions to the reactions obtained by turning, in 753 children, and whether deafness was partial or total and congenital or acquired. Of the 753 children examined by

caloric tests, 489, or 64.94 per cent, gave a positive response. Of the 489 with positive caloric reactions, 444, or 90.79 per cent, also gave responses to turning tests, and 45, or 9.20 per cent, gave no response to turning. In the latter group, 18, or 30 per cent, had partial deafness, in 13 instances congenital and in 5 acquired; 27 showed total deafness, congenital in 18 and acquired in 9. Of the 264 children giving no

TABLE 17.—*Relation of Caloric Reactions to Other Tests in Seven Hundred and Fifty-Three Children (14 Per Cent)*

Caloric reactions present 489 (64.94%)	Turning reactions present 444 (90.79%)	Normal 224 (50.45%)	Partial deafness 146 (65.18%)	Congenital...	99 (67.79%)
				Acquired....	47 (32.21%)
			Total deafness 78 (34.82%)	Congenital...	60 (75.64%)
				Acquired....	18 (24.36%)
		Subnormal 220 (49.55%)	Partial deafness 128 (58.18%)	Congenital...	77 (60.15%)
				Acquired....	51 (39.85%)
			Total deafness 92 (41.82%)	Congenital...	61 (65.64%)
				Acquired....	31 (34.36%)
	Turning reactions absent 45 (9.21%)		Partial deafness 18 (30%)	Congenital...	13 (72.23%)
				Acquired....	5 (27.77%)
			Total deafness 27 (70%)	Congenital...	18 (66.66%)
				Acquired....	9 (33.34%)
Caloric reactions absent 264 (35.06%)	Turning reactions present 19 (7.2%)	Normal 9 (47.37%)	Partial deafness 7 (77.77%)	Congenital...	5 (71.42%)
				Acquired....	2 (28.68%)
			Total deafness 2 (22.23%)	Congenital...	2 (100.00%)
				Acquired....	0
		Subnormal 10 (52.63%)	Partial deafness 4 (40%)	Congenital...	3 (75.00%)
				Acquired....	1 (25.00%)
			Total deafness 6 (60%)	Congenital...	3 (50.00%)
				Acquired....	3 (50.00%)
	Turning reactions absent 245 (92.8%)		Partial deafness 96 (39.2%)	Congenital...	41 (42.70%)
				Acquired....	55 (57.30%)
			Total deafness 149 (60.8%)	Congenital...	61 (40.94%)
				Acquired....	88 (59.06%)

caloric response, 19 were found to respond to the turning tests, 9 of these normally and 10 subnormally. In 245 children (92.8 per cent) turning reactions as well as caloric responses were absent. In the latter group 69 were partially deaf and 149 totally deaf. In both groups cases of acquired deafness slightly outnumbered those of congenital deafness.

Table 18 is constructed to show a somewhat different analysis of the 753 children examined for caloric reactions. We found that caloric reactions were present in both partial and total deafness—in 59.79 per

cent of the partially deaf and in 40.21 per cent of the totally deaf children. These groups are analyzed still further in regard to responses to the turning tests.

Unless one has undertaken to make a careful functional examination of the hearing and the vestibular reactions in a deaf child he can hardly appreciate the time and effort and, I might add, the almost infinite patience required for completing this investigation of 5,348 children. I cannot praise too highly the conscientious, careful and persevering work of those who participated in these examinations extending over many months: Drs. Alice K. Hall, E. W. Hagens and J. W. Holderman

TABLE 18.—*Relation of Caloric Reactions to Other Tests*

Caloric reactions present 490 (65%)	Partial deafness 293 (59.79%)	Congenital 188 (64.16%)	Response to turning present	175 (93.08%)
			Response to turning absent	13 (6.92%)
		Acquired 105 (35.84%)	Response to turning present	99 (94.28%)
			Response to turning absent	6 (5.72%)
	Total deafness 197 (40.21%)	Congenital 140 (71.06%)	Response to turning present	115 (82.15%)
			Response to turning absent	25 (17.85%)
		Acquired 57 (28.94%)	Response to turning present	49 (86.00%)
			Response to turning absent	8 (14.00%)
Caloric reactions absent 263 (35%)	Partial deafness 107 (40.69%)	Congenital 49 (45.79%)	Response to turning present	8 (16.33%)
			Response to turning absent	41 (83.67%)
		Acquired 58 (54.21%)	Response to turning present	3 (5.18%)
			Response to turning absent	55 (94.82%)
	Total deafness 156 (59.31%)	Congenital 65 (41.66%)	Response to turning present	5 (9.23%)
			Response to turning absent	60 (90.77%)
		Acquired 91 (58.34%)	Response to turning present	3 (3.3 %)
			Response to turning absent	88 (96.7 %)

in the examination of the 290 children in the public institutions for the deaf in Chicago; Drs. Hagens, Holderman and R. W. Watkins in the examination of 2,932 children in the public institutions for the deaf in the Middle West, and Drs. D. B. Hayden, Hagens and Watkins in the examination of 2,228 children in the institutions for the deaf in New York, Pennsylvania, New Jersey, North Carolina and Virginia.

SUMMARY

It would not be feasible to undertake a discussion of all the possible bearings which the facts gleaned in this investigation might have on the problems of the deaf. The tables were constructed for the purpose of setting forth these facts clearly in diagrammatic form, so that any one interested in any of these problems might be able to gather the

data he might need. I would call attention to only a few of the outstanding achievements and facts brought out in this investigation:

1. Relation between congenital and acquired deafness.
2. Etiology of acquired deafness.
3. The important rôle played by heredity and consanguinity in congenital deafness.
4. Analysis of congenital deafness, especially regarding the degree of the defect in hearing, whether there are other cases of deafness in the immediate family and the status of the nonhearing part of the internal ear.
5. Analysis of the cases of acquired deafness as to whether the trouble was associated with otitis media, whether the deafness was total or partial and the status of the vestibular reactions.
6. Analysis of cases as to total or partial deafness, whether the condition was congenital or acquired and the status of the vestibular reactions.
7. Analysis of the entire group of cases in their relation to the Schwabach and Rinne tests.
8. Analysis of the entire group showing reactions for the functional hearing tests, for the voice and the audiometer, and the relation of these facts to speech defects.
9. Analysis of the entire group as regards the relation between the vestibular responses (turning reactions) and deafness.
10. Study of 753 cases showing the relations between the caloric tests, turning reactions, defects in hearing and whether deafness was congenital or acquired.

If we have succeeded in throwing new light on these problems of the deaf and have furnished some facts that may stimulate others to further research, leading ultimately to the amelioration of the lot of these unfortunate children, we feel repaid for our efforts.

Clinical Notes

RARE CASE OF ANGIOMA OF THE EAR-DRUM, AUDITORY CANAL, FACE, MOUTH, TONGUE, PHARYNX AND LARYNX *

HENRY P. SCHUGT, M.D., NEW YORK

Nevi angiomatosi are new growths, mostly congenital, and are composed of blood vessels. They are likely to occur any place on the surface of the body. Only four cases of nevus angiomatosus (vinemark) of the ear-drum have been recorded in the literature, and all of these have been continuous with more or less angioma of the external auditory meatus and of the face. In 1889, Wagenhaeuser [1] observed a large angioma which covered the right side of the face and extended into the external auditory meatus and across the ear-drum. Peters [2] presented a similar case at the Otological Society of London, and Haug [3] described a case of nevus angiomatosus of the external auditory meatus and of the upper posterior quadrant of the ear-drum. In the case reported by Haug, the color of the angioma became much darker when the intracranial pressure was increased. The fourth similar case was recently presented at the Otological Division of the Koenigliche Gesellschaft fuer Aerzte of Budapest by Krepuska.[4] In the case presented, there was an angioma on the left side of the face extending toward the lobe of the ear, continuing along the posterior wall of the external auditory meatus and involving the ear-drum.

REPORT OF CASE

The case which I report belongs to the group of vascular tumors. The patient consulted me because of hoarseness of two months' duration. Prior to this time, his voice had been clear. A diagnosis of laryngeal tuberculosis secondary to advanced pulmonary tuberculosis was made. The larynx showed marked swelling of the epiglottis and the interarytenoid space, with deep ulcerations of the posterior wall. The left side of the face and neck was covered with a nevus of reddish-violet appearance. The nevus likewise included the entire auricle, extended along the posterior wall of the canal of the ear, and around the periphery of the drum and stretched across the drum along the malleus (fig. 1). The central part of the drum was not affected, and the light reflex was definitely visible. Hearing was normal. The angiomatous changes, which had existed since birth and, in the patient's opinion, did not cause any complaints, extended from the outer skin over

* Submitted for publication, March 4, 1930.

1. Wagenhaeuser: Naevus cutaneus vinosus, Arch. f. Ohrenh. **27**:162, 1889.

2. Peters, quoted by Fischer, Joseph: Haemangiektatischer Tumor des Trommelfelles, Ztschr. f. Hals-, Nasen- u. Ohrenh. **5**:221, 1923.

3. Haug, R.: Naevus cutaneus des Meatus und des Trommelfelles, Beitr. z. Ohrenh., 1905, p. 183.

4. Krepuska: Trommelfell Naevus, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **2**:872, 1927-1928.

Fig. 1.—Left ear-drum and external auditory meatus. The angioma extends from the posterior wall of the external auditory canal to and on the ear-drum.

Fig. 2.—Angiomatous changes, extending from the outer skin over the buccal mucous membrane and across the left half of the tongue.

the buccal mucous membrane and across the left half of the tongue (fig. 2). The latter was enormously swollen, and the condition could be regarded as a macroglossia. It was bright red over the swollen area, and it filled almost the entire left side of the cavity of the mouth. Similar changes existed on both sides of the soft and hard palate, without, however, touching the median line. On the left side, the nevus extended from the palate down the anterior and posterior pharyngeal pillars to the epiglottis, the left half of which was bright red, so that one had to assume that this part of the larynx was also involved. Since the rest of the larynx was tuberculous, it could not be definitely determined whether or not the angiomatous changes had affected the inner part of the larynx also. Tracheoscopy and esophagoscopy showed the trachea and esophagus to be normal. While the skin of the face, neck, auricle and external auditory meatus was markedly knobby and uneven, with wart formation in places, the changes on the ear-drum and in the region of the mouth, tongue, pharynx and larynx were smooth, and there were no wartlike elevations above the level of the mucous membrane.

COMMENT

This case is described chiefly because of the rare observations in the ear-drum. It is likewise interesting on account of the extensive changes in the mouth, on the tongue and pharynx and in the larynx. The patient died of pulmonary tuberculosis, and, unfortunately, permission for autopsy was not granted by the relatives.

INFECTION INVOLVING THE PHARYNGO-MAXILLARY SPACE

Report of Two Cases *

J. WARREN WHITE, M.D., NORFOLK, VA.

"The pharyngo-maxillary fossa is funnel-shaped with its base up and point down and is of very considerable size. It is the pathway of the great vessels and nerves of the neck. It is the pathway, also, of the vessels to and from the tonsil. The base of the funnel is the base of the skull; the tip is opposite the lower limit of the angle of the jaw. The carotid sheath emerges at the tip of the funnel and for practical purposes, continues as the fossa through the neck to the thorax. The internal boundary of the fossa is the superior constrictor muscle on which the tonsil rests. The external is the inner surface of the ascending ramus of the jaw covered by the internal pterygoid muscle. Superiorly, the inner prolongation of the parotid gland makes a part of the external boundary. The upper cervical vertebrae covered by the prevertebral muscles make a posterior boundary. In the posterior chamber near the median line on the body of the axis there is a lymph node which drains the nose and the upper pharynx. It is this node which suppurates in retropharyngeal abscess."

The quotation in the foregoing paragraph is Mosher's description of the pharyngo-maxillary fossa, and it will help to give a more intelligent idea of how

* Submitted for publication, March 22, 1930.

infection travels from the tonsils to the base of the brain. I submit brief reports of two cases that are unusual and that illustrate the type of clinical phenomenon associated with infection in this region.

CASE 1.—A man, aged 28, seen in November, 1923, stated that he had had but a single previous attack of tonsillitis, which occurred in 1916 and lasted for two or three days. The present illness had developed the day before he was seen by me with a slight chill, elevation of temperature, and pain on swallowing. The temperature was 102 F., and the pulse rate 100. His complaint was stiffness of the jaw and great pain on attempting to talk or swallow. Examination revealed acute tonsillitis with marked swelling of the right tonsil. The following day an incision was made through the anterior pillar, which was followed by a free discharge of pus. The incision was reopened the next day. On December 2, a peritonsillar infection developed on the left side. A similar incision was made and was followed by drainage. On December 4, the infection about the right tonsillar area increased, and a swelling developed at the angle of the jaw. The patient could swallow only with great difficulty, and his temperature was 103 F. A second incision was made through the anterior pillar. The patient seemed to be better and could swallow with less pain, but the swelling at the angle of the jaw increased. On December 6, a temperature of 105.3 F. developed. There was no rigidity of the neck or Kernig sign to indicate a possible meningitis. The swelling at the angle of the jaw was considerable, and an examination of the throat was very unsatisfactory owing to the difficulty in opening the mouth. The same day a spinal puncture was made, and 50 cc. of turbid fluid was withdrawn. Antistreptococcus serum was injected intraspinally. On December 7, the symptoms of meningitis became marked, and the patient died. The spinal fluid contained many pus cells, and the streptococcus was obtained on culture. A culture of the blood was negative, and a culture from the throat showed staphylococci. Autopsy was not performed.

It seemed apparent that there was a direct extension of infection from the peritonsillar tissue to the base of the cranium. The experience in this case seemed to indicate that in an infection of the pharyngo-maxillary space, the condition should always be attacked by an external incision from the tip of the mastoid to the angle of the lower jaw along the anterior border of the sternomastoid muscle, as the only measure that is likely to prevent development of an intracranial complication — meningitis.

CASE 2.—A woman, aged 30, had her tonsils removed because of recurring attacks of acute tonsillitis and definite evidences of a chronic tonsil infection. The following day a chill with a temperature of 103 F. developed. She seemed to be desperately ill. A swelling developed on the right side of the face and neck. There was no recession in the temperature. The pharyngo-maxillary spaces became more swollen. On the third day after the removal of the tonsils, an incision was made externally on the anterior border of the sternomastoid muscle, from the tip of the mastoid to the angle of the lower jaw. A deep-seated abscess was located; the opening communicated with the pharynx. The incision was kept open with rubber drains and irrigated daily, the fluid passing through into the throat. In two weeks the patient was able to leave the hospital, the neck being practically healed.

I believe that the infection was due to the fact that a hypodermic needle accidentally penetrated the fascia and muscular layer at the time of the tonsillectomy, separating the peritonsillar space from the pharyngo-maxillary space.

Abstracts from Current Literature

Ear

THE NONSURGICAL DRY TREATMENT OF CHRONIC SUPPURATIVE OTITIS WITH IODIN POWDER (SULZBERGER). M. D. LEDERMAN, Laryngoscope **40**:397 (June) 1930.

While radical surgical intervention is definitely indicated in certain chronic suppurative conditions of the ear, deliberate and conscientious consideration should be given to those suffering from such involvement and an attempt made to conserve as much auditory function as possible. It is highly essential that those cases definitely requiring surgical intervention be differentiated from those that may clear up by conservative treatment. Frequently, such differentiation is impossible until after conservative treatment has been tried for a longer or a shorter time. The author has found it necessary to employ radical mastoid surgery, but rarely since instituting the use of the so-called dry treatment with iodine powder (Sulzberger). The preparation is an intimate association of iodine with boric acid, being prepared by mixing an iodine solution of desired strength with dry, finely powdered boric acid and evaporating the solvent. This results in an impalpable powder of boric acid finely impregnated with the desired amount of iodine. The action is due to the iodine, which, as the boric acid dissolves in the secretions, is liberated and spreads over and penetrates deeply into the tissues. A prompt osmosis occurs, which permits the iodine vapor to reach areas that instrumentation never touches. Consequently, there is an active germicide present which exerts its power as long as there is any powder remaining in the diseased cavity.

The technic in administering the treatment consists first in thoroughly cleansing and drying the external auditory canal and, as far as possible, the middle ear. Much care must be given to the insertion of small cotton-wound applicators through the perforation into the various parts of the tympanum. No water or irrigating solutions may be used at any time. Gentle suction through a small slender cannula may be employed to remove secretions from the middle ear. The canal and tympanum are then cleansed with 95 per cent alcohol and dried. The final procedure is to insufflate the iodine powder, by means of a hard rubber insufflator, into the tympanum through the perforation. A 0.75 per cent iodine preparation is used in the beginning, the strength being increased gradually up to 2 or even 5 per cent. The treatments are sometimes given daily, but more often from two to three times a week.

SNAPP, Grand Rapids, Mich.

RESULTS OF SURGERY AND OF SURGICAL DIATHERMY IN THE TREATMENT OF CANCER OF THE AURICLE. A. GIESE, Folia oto-laryng. **19**:414 (July) 1930.

Giese reported six cases of cancer of the auricle. In all instances, microscopic examination of excised specimens revealed a carcinoma originating from the basal cells of the epidermis. Three patients received surgical treatment, and the remaining three underwent surgical diathermy. The author found that there is no essential difference between the results of ordinary surgical intervention and those of surgical diathermy, even in the cosmetic results. The most advantageous method has to be decided in each case. Observations on one patient proved that electrocoagulation does not prevent relapses. After-examinations of the other patients showed that operative treatment for cancer of the auricle is a valuable therapeutic method. In two patients with metastases in the regional lymph nodes the observation time was almost four years. The results may therefore be considered as permanent. However, an absolute favorable prognosis cannot be made even in cases in which the operation is performed during the earlier stages.

On the other hand, there is no cause to neglect operative treatment; on the contrary, the experiences of the author show that active surgical therapy may bring good results even in advanced and apparently hopeless cases.

IMPROVEMENT OF HEARING BY MEANS OF OPERATIVE TREATMENT OF ADHESIONS IN THE MIDDLE EAR FOLLOWING CHRONIC SUPPURATIONS. K. MATHÉ, Folia oto-laryng. **19**:424 (July) 1930.

Mathé states that at the clinic in Königsberg operations were performed on patients with adhesions between the handle of the malleus and the promontory of the tympanum. He gives the clinical histories of four patients. After conservative methods had failed, the handle of the malleus was separated from the promontory. In two instances an improvement in the hearing resulted from the operation; in the other two cases, the treatment was not effective. A favorable influence on the hearing could be noted only in cases in which mobility of all the auditory ossicles could be effected. That was possible when only the handle of the malleus was fixed. By separating it from the promontory, the crus of the incus was drawn forward and thus the stapes likewise became free. However, if the body of the malleus is involved in the adhesions, the operation is not effective.

EDITOR'S ABSTRACTS.

Pharynx

ABSCESS IN THE RECESSUS PHARYNGEUS WITH THROMBOSIS OF THE CAVERNOUS SINUS. O. KAHLER, Folia oto-laryng. **19**:402 (July) 1930.

Kahler describes the case of a woman, aged 32. Eight days before the patient was brought to the clinic she became ill with chills and fever. Before that, slight pharyngeal pains had existed. At the time of examination she complained of severe pain in the occiput which radiated to the right shoulder. The pains became more severe when the head was moved. Meningitis and encephalitis were thought of, but a definite diagnosis could not be made. The patient died several days later. Necropsy revealed a retropharyngeal abscess, which presumably started in the lymphatic tissues of the recessus pharyngeus, ichorous arthritis of the atlanto-occipital joint, ichorous thrombosis of the cavernous sinus, suppurative osteomyelitis of the pars basilaris of the occiput and of the right pyramid, ichorous thrombosis of the left vena ophthalmica with following orbital phlegmon and suppurative meningitis directly posterior to the optic chiasm. The author further describes the specimen of the abscess. In the conclusion he points out that careful examination of the nasopharyngeal cavity perhaps would have revealed the abscess in the recessus pharyngeus and incision might have prevented fatal complications. He therefore recommends a careful examination of the nasopharyngeal cavity in all septic diseases.

EDITOR'S ABSTRACT.

Nose

TREATMENT OF MAXILLARY SINUSITIS. JAMES J. KING, Ann. Otol. Rhin. & Laryng. **39**:184 (March) 1930.

In discussing this subject the author divides it into the treatment for acute, traumatic, chronic and malignant maxillary sinusitis. In taking up certain general considerations, it may be mentioned that a warm dry climate is practically always beneficial to patients with subacute or chronic sinusitis. An abundance of fresh air and sunshine is always important, and consideration should be given to the diet. Cold air, cold water or chilling of the body is to be avoided. Swimming should be prohibited. All foci of infection, especially infected teeth, should be removed. In cases of acute suppuration of the antrum, the author irrigates daily through the inferior meatus with a physiologic solution of sodium chloride. It is sometimes necessary to shrink the tissues about the middle meatus in order to get a free

return flow. In the cases resulting from infected teeth, the practice of breaking into the antrum through the alveolar process is condemned. In case a fistula results following the extraction of a tooth, this must be closed and irrigations instituted through the nose.

Traumatic infections of the antrum usually follow a blow or injury to the cheek. The antrum may become filled with blood, and later there may be suppuration. Such conditions usually clear up following a few irrigations.

The author makes a plea for conservative measures in chronic maxillary sinusitis. A window operation through the inferior meatus, giving ventilation and drainage, has been highly satisfactory in the treatment of nearly all patients with chronic cases, with the exception of those with polypi, bony necrosis or new growths. For the small percentage of patients with chronic cases who do not recover with the window resection, the Caldwell-Luc operation or some modification thereof is recommended. Extensive removal of the growth, followed by the use of radium, is necessary in all cases of malignancy.

THE RELIEF OF NASAL OBSTRUCTION IN CHILDREN DUE TO DEVIATION OF THE SEPTUM. WILLIAM WESLEY CARTER, Ann. Otol. Rhin. & Laryng. **39**:199 (March) 1930.

It has always been generally believed that an extensive operation on the nasal septum at an early age may interfere with the normal development of the nose, thus resulting in more or less deformity in later life. For this reason children are often allowed to suffer with a nasal obstruction until they have reached an age when it is felt the operation can be done safely. In order to overcome this difficulty and to give these children relief in early life, the author has devised an operation on the septum which, in his experience, has proved to be satisfactory. Two important factors must be kept in mind in such an operation: (1) the importance of preserving the anterior edge of the septum, as this is the support of the tip of the nose, and (2) the avoidance of dislodging the upper edge of the cartilaginous septum from its position between the lateral cartilages, for here it constitutes the keystone of the cartilaginous bridge of the nose.

The technic of the operation is described by the author in considerable detail. Briefly, it consists in making the usual submucous incision and elevating the mucous membrane and perichondrium from the convex side. Two parallel incisions are then made from behind forward on either side of the apex of the deflection. These incisions are made through the cartilage, but not through the perichondrium. The very narrow strip of cartilage lying between the foregoing incisions and constituting the apex of the deflection is then removed, thus permitting the edges of the upper and lower segments of cartilage to fall together. An incision is then made through the upper segment, parallel with the dorsum of the nose and extending the entire length of the deflection, and a corresponding one made below, parallel with the floor of the nose. The purpose of these incisions is to destroy the resiliency of the cartilage. The septum now consists of an upper and lower ribbon-like segment of cartilage and also bone if the vomer and the vertical plate of the ethmoid are involved, between which is a slight interval corresponding to the apex of the deflection. Both of these segments remain attached to the perichondrium on the concave side. Molding forceps are then inserted, one blade on either side of the septum, and the structure lined up in its normal, vertical position. A gold wire splint is then introduced into either nasal cavity to hold the septum in its correct position.

PERSISTENT NASAL HEMORRHAGE TREATED WITH RADIUM. J. COLEMAN SCAL, Laryngoscope **40**:271 (April) 1930.

The bleeding point in practically 90 per cent of all cases of nasal hemorrhage is located in the anterior lower part of the septum, known as Kiesselbach's area. This area is the site of the junction of the superior maxillary artery and the ascending branch of the palatine artery with its numerous communicating branches.

These form a plexus which bleeds on the slightest irritation or manipulation as the mucous membrane in this region is very thin and the vessels are superficial. In cases of repeated and persistent hemorrhage the author has found the use of radium to be beneficial. The radium, of course, cannot be applied in the presence of free bleeding. This can usually be controlled by means of epinephrine packs and cauterization with a chronic acid bead fused on a metal applicator, but constant packing and cauterization are harmful. The radium produces fibrosis of the mucous membrane and also obliterates the blood vessels. The technic of application consists in the insertion of a 25 mg. capsule of radium, after cocainization of the mucous membrane, the radium being screened first with platinum and then with brass and covered with gutta-percha tissue. It is placed opposite the bleeding area and kept there from three to four hours, the treatment being repeated in from two to four weeks. It was rarely necessary to give more than two or three treatments, and in more than 100 patients treated, no recurrence occurred over a period of five years.

SNAPP, Grand Rapids, Mich.

DIAGNOSIS OF LATENT SEROUS INFLAMMATIONS OF THE ACCESSORY NASAL SINUSES. VOCK, Folia oto-laryng. **19**:396 (July) 1930.

According to Vock, chronic serous inflammation generally affects the maxillary sinus. In most instances it can be diagnosed with the aid of the usual diagnostic methods, such as median rhinoscopy, puncture, irrigation, illumination and simple roentgenoscopy. However, sometimes all these aids fail, and a swelling of the mucous membrane in the sinuses is not detected. Other diagnostic measures, such as exploration of the maxillary sinus by means of a sound or Mühlenkamp's method of injection of a solution with following aspiration, are likewise not absolutely reliable. According to newer experiences, the most reliable diagnostic method in cases of suspected serous inflammation of the accessory nasal sinuses is roentgenoscopy after injection of a contrast medium. The 20 per cent iodized sesame oil is used in preference to the 40 per cent solution. This contrast medium is injected while the patient's head is inclined slightly forward and toward the side affected. When roentgenoscopy is done, the patient should be in a sitting position. This method of roentgen examination following the injection of contrast material is especially valuable in latent cases, when the nose does not give indications for serous inflammations and when the patient is troubled with headaches and with a feeling of pressure in the regions of the superior maxilla and of the temple and with catarrhs of the respiratory passages. The contrast medium is injected following puncture and irrigation. In many of the latent cases the irrigation fluid is clear and the illumination does not reveal changes; however, roentgenoscopy following the injection of contrast material shows the pathologic conditions. Filling with contrast material is contraindicated when pus is detected during the irrigation.

INDICATIONS AND CONTRAINDICATIONS FOR RESECTION OF THE NASAL SEPTUM. H. RICHTER, Folia oto-laryng. **19**:399 (July) 1930.

Richter emphasizes that it is necessary to consider all etiologic factors before performing resection of the nasal septum. This operation is not without danger and yet it is often done without sufficient cause. The author observed many patients who came to the clinic after a resection of the nasal septum had not brought the desired results. In discussing deformity of the nasal septum as a cause of difficult nasal breathing, the author stresses that resection should not be done unless it is absolutely sure that the deformed septum is the obstructing factor. It has been observed that in a large percentage of patients with difficult nasal breathing only the removal of hyperplastic parts of the nasal conchae, especially of the posterior terminations of the lower conchae, is effective. This was noted also in patients who had previously undergone submucous resection of the nasal septum. In cases of vasomotor rhinitis caustic treatment of the swollen conchae is usually effective.

Resection of the nasal septum is necessary only in rare cases of vasomotor rhinitis. The author thinks that a considerable number of resections would not be performed if the other parts of the nose that might obstruct the nasal air passage were more carefully examined. A deformity of the nasal septum is rarely the only cause of an inflammation of the air passages or of a disorder of the middle ear. In many instances catarrhs are caused by abnormal conditions of the mucous membrane. In cases in which difficult nasal breathing causes pulmonary or cardiovascular disorders, resection of the nasal septum is sometimes helpful, and the same is true in asthma and in reflex neuroses that are caused by difficult nasal breathing. Resection is not advisable in hemorrhages of Kiesselbach's area. In cases of suppurations of the frontal sinus and of the ethmoid sinus, a submucous resection of the nasal septum is indicated only when the deviation is so marked that irrigation of the frontal sinus and therapeutic intervention in the ethmoid sinus are otherwise impossible, or when the deformity of the septum causes retention in the chronic forms of these suppurations.

APPARATUS FOR NASAL TAMPONADE PERMITTING NASAL BREATHING: RHINOTAMPONATOR. N. ÉRCZY, Folia oto-laryng. **19**:430 (July) 1930.

Érczy points out that the ordinary nasal tamponade makes nasal breathing impossible and thus leads to undesirable conditions. Attempts to employ inflatable balloons for nasal tamponade go back several decades. However, these methods do not permit nasal breathing. The apparatus constructed by the author consists of a rubber balloon which is dimensioned according to the shape and size of the nose. A rubber tube that goes through the balloon serves as a conveyer for the air breathed in. In the forepart of the balloon there is also a small rubber tube by means of which inflation is done. This tube is closed air tight by a special device. The author describes how the apparatus is used. He employed it in thirty-seven cases after surgical intervention and in nine cases of spontaneous nosebleed.

TREATMENT OF OZENA BY MEANS OF LOCAL IMMUNIZATION WITH POLYVALENT AUTOVACCINE FROM KAPSELDIPLOBACILLI. W. JELIN, Folia oto-laryng. **19**: 434 (July) 1930.

The therapeutic method recommended by Jelin is based on researches on kapseldiplobacilli. It had been found that in patients with ozena the nasal mucous membrane contains not only one certain type of kapseldiplobacilli but several types, and also that the flora of kapseldiplobacilli varies in different patients. The first requirement of the treatment prescribed by the author is that the types of kapseldiplococci are determined (behavior toward various sugars). A polyvalent vaccine is prepared from these microbes. A tamponade is made with strips of gauze. For each application from 3 to 5 cc. of vaccine is used. The tamponade is made every day and should remain during the night. Patients usually require from thirty to thirty-five applications. In some instances a second series of applications had to be given after the treatment had been interrupted for thirty or forty-five days. The author employed this treatment in twenty cases; good results were obtained in nearly all instances. The local immunization treatment was not effective in the case of a girl, aged 9, in whom ozena had begun in the second year of life. In the other patients, however, the fetor disappeared and the scab formation was also reduced. The patients were observed from six to twelve months, and the fetor did not return. Occasionally, scabs formed again, but disappeared quickly. In some instances a regeneration of the nasal mucous membrane could be noted. The author recommends the further investigation of this treatment.

DIRECT EXAMINATION OF THE MAXILLARY SINUS BY MEANS OF ENDOSCOPY. M. SLOBODNIK, Folia oto-laryng. **19**:437 (July) 1930.

Slobodnik describes his method as follows: The medial wall of the maxillary sinus is pierced by means of a trocar. Then the mandrin is removed and the

endoscope is introduced through the tube. By moving the endoscope the maxillary sinus can be surveyed. If necessary, the sinus may be irrigated. In this case, the tube of the trocar is introduced a little further and the endoscope is removed. Endoscopic examination of the sinus should be preceded by repeated cocainization of the lower nasal duct. A few drops of a solution of epinephrine may be added. The author further describes the changes that may be detected with the aid of the endoscope. In the conclusion he stresses the following advantages: 1. The picture of the mucous membrane of the maxillary sinus may be observed during the first consultation. 2. By repeated examinations and irrigations it can be determined whether the pathologic conditions in the sinus have improved. 3. During nasal surgical intervention a survey of the maxillary sinus is possible.

EDITOR'S ABSTRACTS.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Monthly Meeting, Feb. 3, 1930

SAMUEL SALINGER, M.D., *President*

NEUROSIS AS A CAUSE OF SPEECH DISORDER. DR. ROBERT WEST, University of Wisconsin (by invitation).

Although one cannot say that neurosis is responsible for more disorders of speech than all other causes, it appears to be to blame for more disturbances of speech than any other single group of causative conditions. The chief reason for the prominence of neurosis as a pathogenic factor in the function of speech is to be found in the emotional coloration of neurosis.

The neurotic patient is unable to (or at least does not) exert the normal cerebral inhibition on the centers where emotional movements are subtended, viz., the thalamus and striate bodies. These emotional movements are not engaged in generally by all of the muscles of the body, but are more or less concentrated in the muscles of respiration, mastication and deglutition and in the superficial facial muscles. The only muscles of the groups mentioned that do not play a direct part in production of speech are a few facial muscles around the eyes and across the forehead. The use of these groups of muscles for the enacting of their intended emotional rôles does not always preclude their playing their learned speech rôles. High emotional excitement of any color robs us of the power of skilful and delicate articulation, and certain emotional states block speech entirely. The emotional states most disastrous to good speech are those the inherited pattern of which involves muscle movements opposite in direction to, or greater in intensity than, the movements necessary for speech. Many a case of stammering begins with what seems to be a sobbing mechanism. Other emotional patterns, such as those of fear and anxiety, do not so much involve actual movements conflicting with those necessary for speech as they preempt the muscle groups for other uses by setting them in a state of hypertonicity. The normal functioning of speech requires many movements of extremely light touch. Only the explosives can be made with intense muscle contraction. Other sounds require delicate poise between antagonistic muscles. Any neuroses, therefore, characterized by fear or anxiety tend to interfere with speech by disturbing this poise.

Cases of aphonia for which no possible contributory cause could be found were studied by means of the x-ray. The neurosis seemed the only explanation of the loss of voice. I found that it was impossible to analyze the roentgenograms until there had been developed a series of pictures of subjects whose voice training gave them the ability to hold the laryngeal adjustments in various degrees and combinations of tension.

In certain cases, at least, of hysteric aphonia, the neurosis shows itself in a hypertension in the direction of phonation but beyond the point of efficient production of voice. It is this principle of overtension that is a frequent neurotic cause of disorder of speech. It is quite possible that if one talked with one's hands, disorders of speech would be less frequent.

DISCUSSION

DR. ELMER L. KENYON: Dr. West's method of approach to the etiology of disorders of speech is relatively new and not familiar to me. In using the term "functional aphonia" meaning the production of voice by action of the mechanism

of speech, in which the normal loud quality is totally replaced by a quality closely resembling a whisper, one should bear in mind that the mechanism of production of speech acts normally to produce each of these qualities. I have studied closely the action of the peripheral mechanism of speech in hysteric aphonia, and feel convinced that its action was definitely that of the normal whispered voice. If this be true, the nervous disturbance is one that temporarily and functionally destroys the possibility of producing action of the mechanism of speech for the loud quality of voice, but does not interfere with normal innervation for the whispered voice. I feel that such a disturbance probably had its seat in the cortex, and that it was produced by a normal innervation and muscular action of the speech mechanism for the whispered voice. That such a voice could be imitated by artificially producing wrong muscle action seems to me possible; but would this method of study throw light on the fundamental neurotic disturbance? At this moment it seems to me that wrongness of muscular action in stammering, in aphonia and in nasality could more probably be explained on grounds quite different from those suggested by the speaker. But it might be that certain muscular disturbances of various disorders of speech could be explained, at least in part, by the neurotic tendencies suggested by Dr. West, and his point of view should be carefully studied.

DR. MEYER SOLOMON (by invitation): Many different terms are used in describing neuroses; here it has been used as a bodily disturbance due to a psychologic cause. In respect to disturbance of speech the mind can affect the body in two ways: by volition and through emotion. By volition it can affect the musculature of the extremities, of the trunk and of those parts of the body under volitional control. By emotion it can affect any portion of the body, even the chemistry of the blood, through fear, anger, etc. One may have disorders of speech due directly to volitional control and others due to emotional disturbance. The first condition is typical of ordinary hysteria. In this condition, things occur owing to the person's idea that he or she is unable to do certain things. If convinced that one is unable to do a certain thing, no effort will be made to do it, and every effort will be made to do everything except that thing. The hysterical person cannot do anything with the body that a normal person cannot do. A malingerer may purposely, consciously, imitate loss of power of some sort. The malingerer knows he can do the things he claims he cannot do, and the hysterical person really believes that he cannot. In hysteria, all that can happen to the mechanism of speech is that the patient either makes no effort to use the proper muscles or makes the wrong effort.

In the emotional disturbances of speech, such as stuttering, there can be any sort of upset in the peripheral speech machinery. It is not the same as hysteria. It is not a purely voluntary state. In stuttering one of the two main things that happens with the peripheral speech machinery is that the person in his haste produces an attitude in which he is unable to produce any sound at all, or he is able to produce the first sound but is unable to advance to the second one.

As the speaker saw the problem, the speech machinery can be upset in stuttering and allied emotional disturbances or in hysteria which is due to autosuggestion. In the latter, either no effort is made or the wrong effort. Among the wrong efforts is the hypertension that Dr. West has brought out. The idea of utilizing the roentgen rays in this work was new to me, but no difference what the observations are, in the speech machinery one must relate it back to the general psychologic state of the person.

DR. AUSTIN A. HAYDEN: I wish to call attention to the fact that the last speaker at one time stuttered severely, and the fact that he had completely overcome it was a great achievement and a splendid tribute to the men who work on disorders of speech. The general otolaryngologists, I think, should be very proud of having among their number men like Dr. Kenyon who devote their entire time to the remedial treatment for these distressing disorders. The work is very tedious, and often it is a labor of love rather than one of remuneration. In my

opinion, all physicians in general practice need to know of disorders of speech is the name, address and office hours of a man who specializes in the treatment of patients with such disorders. All patients with defects of speech should be referred to these specialists instead of being operated on first to cure them of the defect.

I am greatly impressed with the presentation of Dr. West, but thought his points would have been carried much better had he plotted the roentgen observations against the larynx of the normal person.

DR. ROBERT WEST: Referring to the use of the phrase "hysterical aphonia," I am not entirely satisfied with the term "hysterical" in this connection, and I used it not because it is accurate, but because it is the one used in the literature to describe the condition in which aphonia appears without definite structural or physiologic cause. I would define the condition as a neurosis, and the more I work with patients with defects of speech, the more I am inclined to think of a neurosis, not as something that is caused by an unfortunate incident in the patient's life, but as a condition resulting from a whole complex of situations that affect the neurology and physiology. One of my students, for example, who is at present working on an analysis of the blood and urine of stutterers, has found a very high dextrose content of the blood and a high carbon dioxide content of the urine.

I agree that many of these patients get well after psychoanalysis, if one could find the disturbing complexes, such, for example, as one that develops because a boy has a marked enuresis over a period of years. The embarrassment that comes from this might cause a defect of speech, but I believe that if this occurred it was due to something more than the embarrassment. I am attempting to learn what the connecting link is; whether it comes through the central nervous system, the blood or some other channel.

As to why some patients can make a consonant sound but cannot go on to the vowel sound, I believe the consonant sound can be made because it is an explosive and the vowel sound is not. The explosive sounds can be made much more easily than the half position necessary for the vowel sound. The neurotic person seems to have difficulty in leaving a contracted position and going at once to a muscle position in which there is normal muscle balance which allows for phonation. There would be fewer disorders of speech if we talked with our hands, because the hands are not connected with the emotional centers which control the mechanism of speech.

I believe one could say that hysterical speech difficulty may be outside of cerebral direction but not outside of brain direction, certainly not outside of central direction; quite possibly it is influenced by many factors of muscle metabolism.

FOREIGN BODIES IN THE NOSE. DR. WILLIAM A. SMILEY.

Most cases of foreign body in the nose occur in children. Beans, matches, cotton, corks, buttons, tin toys and pencil erasers, being among the most frequently used objects, are readily accessible, appropriate for insertion, and consequently frequently seen by the physician. Usually, the child is frightened by having put some article into the nose and tells the parents, or if too young to talk calls their attention to it by crying, pricking at or rubbing the nose. A large percentage of foreign bodies are removed by the parents immediately after insertion. When they are unable to remove them, they usually call for aid at once. However, there are a few cases in which the parents never suspect the presence of a foreign body in the nose, but consult a physician because of some symptom resulting from the irritation of the particular object.

The most frequent symptom is unilateral nasal discharge. The discharge is usually mucopurulent, but often serous. At times there is a history of bloody expectoration. Nasal obstruction on the affected side is usually present. General symptoms are commonly absent, and when present are due most frequently to

the swallowing of pus or blood which drips backward into the throat. Loss of appetite and loss of weight may be present. Secondary anemia was marked in one of the essayist's cases.

A careful examination is necessary in every case in which there is unilateral nasal discharge. The nose should be carefully cocainized, and if the desired shrinkage of the mucosa does not result, epinephrine should be used. The discharge should be removed carefully, and one can usually see an area where there is some bleeding. This should be carefully palpated with a probe if a foreign body cannot be seen. An object that has been in the nose for any length of time will give the sensation of bare bone. Even cotton will become incrusted with a calcareous deposit and yield this sensation.

Except in refractory children, general anesthesia is rarely necessary for the removal. Each object will present its own problem in removal, and the method must be decided on in each case.

Careful clinical observation is of the greatest value in arriving at the correct diagnosis and intelligent treatment.

SOME DIAGNOSTIC CONSIDERATIONS WHEN VERTIGO IS A SYMPTOM. DR. LAWRENCE J. LAWSON.

To interpret clinical observations properly it is often necessary to combine practical theoretical considerations with recognized anatomic observations.

REPORT OF CASES

CASE 1.—B. T. B. was admitted to the Evanston Hospital on Jan. 25, 1929. The chief complaint was dizziness. One month previous to admission severe pain suddenly developed on the right side of the head, principally back of the right ear. No infection of the ear was present. Following the initial attacks of pain, there were several attacks of vertigo. There was at no time a loss of consciousness. He was able to reach a chair or bed with little difficulty. The attacks were characterized by blurred vision and extreme vertigo. There was no tendency to fall to a particular side. There was no nausea or vomiting and no recurrence of the headache. Four days before admission he had a severe attack of vertigo while at his desk; he felt quite weak, and his associates stated that he was very pale. There was a daily recurrence of these attacks until admission to the hospital. He had been well until the present attacks. The family history was negative for anything related to the problem presented. A neurologic examination was made by Dr. Bassoe the day after admission. The pupils were equal and regular. The optic disks were normal. The deep and superficial reflexes were normal. Wassermann and Bárány tests, complete rest and discontinuance of tobacco were advised. The Wassermann reaction was negative. The external auditory canals were unobstructed, and the drum membranes were intact and gave no evidences of infection. The second day after admission the vestibular tests were given by caloric stimulation with the patient at rest in bed. Water at 10 C. was used. Irrigation of the right ear with 30 cc. gave a horizontal rotatory nystagmus to the right of normal amplitude, after forty-five seconds, lasting sixty seconds. Irrigation of the left ear with the same amount gave a horizontal rotatory nystagmus to the left of increased amplitude, after thirty seconds, lasting ninety seconds. This was interpreted to mean a noticeable increase in the irritability of the right labyrinth. The patient continued to improve in his freedom from vertigo, had no attacks while under observation, and was sent home after five days to plan an extended rest in Florida. Two days later, he was seized with a violent vertigo at night and died ten minutes after readmission to the hospital. At necropsy there was revealed both recent and organized thrombosis of the basilar and right vertebral arteries.

CASE 2.—Miss P. K. was admitted to the Evanston Hospital on July 9, 1929. This patient had recovered from a bilateral otitis media associated with scarlet

fever, only to be precipitated into another bilateral attack by measles. On admission to the hospital two weeks after the onset of the second attack, the temperature was 105 F. and the leukocyte count was 16,500, of which 78 per cent were polymorphonuclear cells. There was a right acute suppurative mastoiditis, a marked right cervical adenitis and a subsiding left otorrhea. Roentgenograms revealed necrosis of the right mastoid cells but no changes in the left. The right mastoid was opened on the day of admission, the lateral sinus exposed, but found unchanged, and the dura not exposed. Cultures revealed gram-positive cocci in pairs and chains. On the fourth postoperative day, the cervical adenitis appeared to be undergoing suppuration, and an incision was made under gas anesthesia which yielded 20 cc. of pus. On the eighth day there was a distinct phlegmonous state in the right cervical region; under gas anesthesia, three deep incisions were made, but no pus was found. The cervical infection decidedly retarded healing of the mastoid wound. On the twelfth day, pus came from the deep incisions and by the fourteenth day there was a striking local improvement, but the patient looked toxic. The patient had complained of hearing foreign sounds. Hearing in the left ear was somewhat diminished. There was a slight horizontal rotatory nystagmus on looking to the extreme left. Sitting up or sudden turning of the head produced vertigo. The left drum membrane was intact with no bulging, and there were no acute symptoms referred to the left ear. The patellar reflexes were brisk; there was a bilateral ankle clonus, slightly more pronounced on the left at times, a questionable bilateral Babinski sign and no Kernig sign. On the eighteenth day, with local symptoms rapidly clearing, with the temperature, pulse and respiratory rate near normal, the patient looked ill, and symptoms of intracranial pressure became manifest. The spinal fluid revealed 800 cells per cubic millimeter, chiefly polymorphonuclears, and not under noticeably increased pressure. The white blood count was 35,000 of which 76 per cent were polymorphonuclears. Disturbance of coordination, while suggesting cerebellar involvement, did not definitely lateralize the site of lesion. If the predominance of polymorphonuclear cells over lymphocytes in the spinal fluid is taken as an index to poor operative prognosis, this contraindication was present. In the absence of further localizing symptoms and because of the poor condition of the patient, no intracranial exploratory work was done. The patient expired on the twentieth postoperative day. Necropsy revealed normal right-sided postoperative observations. There was no meningitis. On opening of the left temporal bone (the symptomless side), an intact drum membrane was revealed and softening of a few cells high in the petrous portion with an erosion at the internal auditory meatus. There was an abscess stalk from this point of contact to the adjacent cerebellum, the left lobe being filled with pus. There was a localized meningitis and a beginning left sigmoid sinus thrombosis.

Comment.—The patient with the thrombosed basilar and right vertebral arteries had transient extreme vertigo with an experimental nystagmus indicating only a slightly increased irritability of the right side. This discordance and the absence of an acute pathologic process of the ear point to a central lesion. The unsteadiness and tendency to fall during the attacks without falling definitely to the right or left are indicative of cerebellar involvement. The experimental horizontal rotatory nystagmus and the conjugate movements of the eye, with equal pupils, exclude a lesion of the oculomotor brain. An infratentorial lesion was indicated by the extreme vertigo, pallor and faintness. The patient with the cerebellar abscess had a slight tinnitus, increasing deafness and slight nystagmus on looking to the extreme left, which would have merited more attention had not the presence of such an extensive pathologic process on the opposite side and the contraindications to vestibular testing masked the situation. The left vestibular and cochlear nerves were probably blocked by the adjacent necrosis, but the left cerebellar abscess would account for the slight continued left nystagmus. These pathologic developments would emphasize further the urgency of repeated bilateral roentgenologic examination in infections of the ear running an irregular course.

March 3, 1930

SAMUEL SALINGER, M.D., *President, in the Chair*

ABSCESS OF THE BRAIN. DR. G. W. BOOT.

Dr. G. W. Boot presented three patients who had been operated on for abscess of the brain.

MASTOIDITIS? DR. T. C. GALLOWAY.

A woman, aged 23, was admitted to the Cook County Hospital three weeks before presentation with the complaint that her ear had discharged for five years. Two weeks before, there was a severe earache which soon subsided, but during the last few days it recurred, accompanied by vomiting. On admission there were severe headache, marked rigidity of the neck and a slight Kernig sign. The knee jerks were approximately normal. The visual fields and disks were normal. The spinal fluid was under considerable pressure and there were 3,200 cells to the cubic millimeter.

Mastoid operation was performed. The lateral sinus was uncovered and found to be normally bluish. The dura over the tegmen antri was uncovered and seen to be thickened and grayish. Pulsation was not visible but was easily felt.

There was a question as to whether the patient had pia-arachnoiditis, beginning encephalitis, a subdural abscess or localized meningitis which should be drained, or possibly an abscess of the brain. There were no focal symptoms before operation. The reflexes were equal on both sides; there was no nystagmus, no ataxia and no asynergy.

Nothing further than a simple mastoid operation with wide dura exposure was done, and the patient made an uneventful recovery. At once the temperature, which had been 100.5 F., subsided to normal and the discharge from the ear disappeared. Successive spinal fluid cell counts were 900, 50 and 6. The patient felt strong and was apparently well at the time of presentation.

Dr. Galloway recalled three patients from the County Hospital shown three years before with similar symptoms of meningitis and fluid observations, also treated successfully only by wide dural exposure, and he expressed the opinion that patients who have no focal symptoms should be treated conservatively.

Operations have been properly performed by masters of operative technic and diagnostic finesse, who have found definite localized lesions inside the dura which have led them to these operative procedures; but these should not lead the ordinary otologist into rash intervention or invasion through the dura without very positive indications.

ABSCESS OF THE BRAIN. DR. CHARLES F. YERGER.

In J. C., a boy, aged 8, with no previous history of disease of the ear, acute suppurative otitis media (left) developed on April 1, 1929. Because of slight persistent tenderness over the mastoid, a simple mastoid operation without exposure of the dura was performed on April 20, and the patient made a good recovery.

On May 29, the patient complained of attacks of slight nausea and vomiting and had a temperature of 99 F. The possibility of an abscess of the brain suggested itself, but the more frequent condition of gastro-intestinal upset in a child of his age was considered more likely. Treatment along this line was instituted, with good results. A week later the patient again complained of nausea and vomiting and again he was treated for gastro-intestinal disturbance.

Nothing more was heard from the patient until June 13, when Dr. Yerger was called in as a consultant. The child then appeared to be very ill, he was emaciated and anemic and the temperature was 102 F. There were marked stiffness of the neck, a positive Kernig and Brudzinski sign and anomia, the latter being considered pathognomonic of an abscess of the left temporal lobe. The spinal

fluid was under increased pressure and slightly turbid; the cells numbered 3,000, 95 per cent of which were polymorphonuclears. No micro-organisms were found on smear or culture.

A diagnosis of abscess of the left temporosphenoidal lobe, associated with a circumscribed meningitis, was made and an operation was performed on June 17.

The previous mastoid wound was opened, making a radical mastoid operation, with complete removal of the tegmen antri and tympani. The exposed dura was apparently normal and the brain showed no pulsation. A longitudinal incision was made through the dura from before backward.

The brain appeared normal except for the absence of pulsation. A Gifford brain searcher was inserted over the region of the tegmen antri but no pus was found. It was withdrawn and reinserted over the region of the tegmen tympani and creamy yellow pus flowed out in large amounts under considerable pressure.

A one-fourth inch (0.64 cm.) nonperforated drainage tube was inserted into the abscess cavity and the surrounding area packed with iodoform gauze. When the drainage tube ceased to drain it was removed and another inserted, a tube being kept in the abscess cavity for thirty-six days. No irrigation of the abscess was done. On two occasions, aspiration was attempted through the drainage tube but was unsuccessful. Three or four times a medicine dropper full of a 2 per cent solution of mercurochrome-220 soluble was injected through the drainage tube into the abscess cavity, apparently with good results.

Dr. Yerger emphasized two features in this case which he considered factors for safety: 1. One should not operate on an acute abscess of the brain until it has become firmly walled off or encapsulated, which requires from three to four weeks. 2. The drainage tube should be kept in place until the abscess cavity has become obliterated, or until suppuration has ceased. In his opinion, it is less harmful to leave the drainage tube in place too long than to remove it too soon.

DISCUSSION

DR. G. W. BOOT: I congratulate Dr. Galloway on his success. I have on several occasions made a slight opening in the dura and found nothing inside. In one or two cases I thought this was a good thing to do as it might relieve the tension and prevent further necrosis. The patients went on to recovery without the formation of pus.

DR. NOAH SCHOOLMAN: A child, aged 7, was admitted to hospital in a state of coma and no history could be obtained. Sugar was found in the urine and there was a question whether the coma was due to glycosuria, but the child kept her hand to her head and moaned. Examination showed chronic suppurative otitis media with aural polypus. Operation was decided on. The mastoid was opened and a cholesteatoma was uncovered which filled the mastoid cavity and covered a large area of the cerebellum. The dura felt like parchment and had no granulations. Spinal puncture revealed an increased cell count and pus in the fluid. The fluid cleared up within three weeks and the child made a good recovery. She recently returned to the clinic with otitis media of the opposite side which was treated by paracentesis. I agree with Dr. Galloway that it is well not to explore too far in such cases.

DR. J. HOLINGER: In one patient on whom I operated three years ago, I found the dura over the cerebellum under increased tension and incised it. The patient did well for a time and then developed septic attacks with chills and abnormal temperature. The sinus was laid bare extensively, and an extradural abscess was drained. Again he did well for a week. Then the temporal lobe was uncovered and the dura was found not to be pulsating. It was incised and several drops of pus were obtained. The prognosis at that time was poor. In the course of time the brain substance forced itself out of the incision of the dura. For some time (two weeks) this hernia baffled me, but within two days it was covered with good epidermis. The boy now has a hernia about the size of half a walnut, but has no discomfort from it whatever and is doing well.

DECALCIFICATION OF TEMPORAL BONES: THE USE OF X-RAYS FOR DETERMINING WHEN DECALCIFICATION IS COMPLETE. DR. E. W. HAGENS.

This article appeared in full in the July, 1930, issue of the ARCHIVES.

UNLOCKING THE PETROUS PYRAMID. DR. WELLS P. EAGLETON, Newark, N. J.

This paper is to appear in full in a later issue of the ARCHIVES.

DISCUSSION

DR. ROY R. GRINKER (by invitation): I think that Dr. Eagleton's apparent lack of esteem for neurologists is due to the fact that he is a pretty good neurologist himself. He has demonstrated that his knowledge of neurologic anatomy is much better than that of the average neurologist. In the field of abscesses of the brain he has done what Cushing has accomplished in surgical procedure in tumors, namely, the obliteration of outside specialists. This probably is a sign of advancement.

Dr. Eagleton spoke of finding quite early evidence of localized meningitis and that before meningitis secondary to suppurative otitis media becomes general, it may give rise to bloody, but not purulent, fluid. I find it difficult to reconcile that with his knowledge of the wide open subarachnoid space where adhesions form with great difficulty when there is any degree of pressure. It has been my fortune to study several cases of generalized damage of the brain following otitic infection in which the general symptomatology has been serous meningitis plus a toxic reaction of the brain. These patients either died within the first few days following the infection, or if the source of the infection was relieved, they recovered. I wonder whether Dr. Eagleton's examples of exposure of the dura, or penetration of the dura, and later a free flow of fluid, have not been examples of this serous toxic meningo-encephalitis, which would have cleared anyway provided the source of infection had been treated.

I am interested in the frequency with which infections of the nose give rise to brain conditions. A recent article published in England demonstrated how easily nasal fluids may infect the subarachnoid space. Experiments have shown that within two hours after dropping stained fluid into a rabbit's nose it could be distinguished in the meninges at the base of its brain.

I think that Dr. Eagleton's description of the variations in the visual fields are remarkable. It seems to me from the study of certain rather similar transient, varying hemianopias due to an adjacent lesion, a so-called retrolenticular internal capsule softening, in which the optic fibers are not involved directly but by vascular reaction, that the visual fields become normal rapidly. Edema thus can develop and can seriously affect the function of the optic fibers temporarily. In Dr. Eagleton's description of the carotid artery, his decision to attempt only partial ligation seems quite reasonable, for it is frequently found that in the circle of Willis some of the vertebral arteries may be very narrow and not sufficient to allow compensatory circulation to take place, so that a permanent hemiplegia may result.

I am also impressed by the fact that otologists apparently do lumbar punctures without as much trepidation as neurologists have. If neurologists suspect intracranial pressure of any gravity, particularly of posterior fossa origin, they fear the result of a lumbar puncture, and I am surprised that the otologists encounter so little apparent trouble in doing punctures in cases of cerebellar abscess. I have seen patients in whom death occurred within a very few minutes following this procedure, and I feel that a little more caution should be used in doing spinal punctures. In spite of the fact that the information gained is often very important, I hesitate to do spinal punctures in these cases.

DR. ALBERT KNAPP: I am the kind of a neurologist who specializes in the reflexes, and when there is an infection of the middle fossa of the skull, I look for symptoms from the oculomotor nerve, if there is infiltration, or congestion or swelling in the organs in the middle fossa. I do not hope to find these myself,

but I enlist the interest of the physicians and the hospital nurses because the symptoms of the oculomotor nerves are transitory, sometimes lasting only a very few moments, perhaps only half a minute. This happens often in tumors, and I think that I was the first to emphasize these symptoms in tumors, but it happens much more frequently in abscess. The work of McEwan on abscess of the temporal lobe shows that in a surprising number of cases of abscess, there are changes in the pupils and also in the oculomotor nerves.

It has been stated that in some cases of inflammation of this region, it is sufficient to open the skull to afford relief and I have had the same experience. I think that this might be due to the influence of the air as well as to the relief of pressure. Also in some other fields, the influence of air has been surprising. It is known that in the presence of tuberculous disease of the peritoneum if the abdomen is opened the tuberculosis will heal. I have had the same experience in one patient with cancer of the stomach, liver, glands and peritoneum. The inoperable growth subsided for three years after a tentative laparotomy. In diseases of contents of the skull the symptoms often disappear following skull puncture, but only for a short time, in spite of the small opening of the skull.

I have observed two cases in which an encephalitis started apparently with symptoms in the ear, and there are some more cases of this type in the French literature.

When there is an abscess of long standing in the brain I think that it is well to search for focal purulent infection in the body, especially in the lungs after cases of bronchiectasis.

Dr. Alfred Lewy: I think that Dr. Eagleton might have the honor of being recorded as the first man to practice drainage of the cisterna pontis lateralis. When I published my paper on this subject, I found nothing in the literature. I reported some time ago the case of a boy who developed meningitis following a mastoid operation; I enlarged the mastoid wound and went into the cisterna pontis. The boy recovered, but still has vertical nystagmus. There were no other neurologic symptoms that I could discover at this time.

When a collection of fluid accumulates on the petrous pyramid or even in the cisterna pontis lateralis, it is not necessary to destroy the labyrinth to get at it. In my case I went right through, regardless of the posterior canal, but I have since found that one can stop before reaching the posterior canal, and with an ordinary septal elevator can separate the dura from the petrous portion, and break into the cisterna pontis lateralis and find the fluid. The operation may also disclose small abscesses in the posterior fossa in the region of the saccus. I have had no experience in going into the superior surface, as Dr. Eagleton has, and I do not believe the operation that I carried out would reach the marrow space in the tip as Dr. Eagleton described it.

Dr. J. Holinger: It is often forgotten that the symptoms of the brain may change from day to day and even in a few hours. A physician may be called in consultation and fail to find symptoms that have been described as present. Symptoms may disappear and others appear in very short time.

I cannot agree with Dr. Eagleton that the spaces medial of the labyrinth in the tip of the pyramid of the temporal bone are always marrow spaces. The atlas corrosion anatomy of Siebenmann shows pictures of temporal bones that have been filled with Wood's metal by pouring the metal into the cells of the mastoid. They showed also that there is a connection between the mastoid cells and the spaces in the tip of the pyramid. I think that evidently both views are correct; there are cases in which there is no connection and others in which there is a direct connection with the cells. This connection may be formed by a canal which goes along the edge of the pyramid. Regular air cells in the tip of the pyramid are an exception, if not a rarity.

Dr. E. W. Hagens: In the specimens that I have studied I searched for pneumatic spaces in the front end of the petrous bone, but the only spaces that I have seen so far have been marrow spaces, as Dr. Eagleton said. They are

filled with marrow cells. I have, however, seen pneumatic spaces lateral to the eustachian tube region. The cases studied were limited and I speak only from personal experience.

DR. WELLS P. EAGLETON: I am not a pathologist and do not believe that all of the future of medicine lies in pathology. When I studied the microscopic specimens in my three cases, the sections of the petrous apex looked very different from those of the many mastoids which I have seen. The petrous apex is developed from one center, which appears to me to be the explanation of the observations. The specimens were different and it was also an amazing clinical fact, that in examining necropsy material many cases showed nothing but redness or discoloration of the bone, without caries. I will get a good pathologist to substantiate Dr. Hagens, for I know these cases were different and that there are two kinds of bone in the temporal.

In reply to Dr. Holinger, I agree that when dealing with infection, the symptoms are associated with edema and change rapidly, but edema of the brain is different from that of other parts because when it spreads it gives marked symptoms. A patient with an abscess of the temporal lobe at one moment will show symptoms and the next moment will not. This is especially the case with the ocular field, because the factors are due to edema.

Regarding Siebenmann's work, I urge any one who can pick up a volume of his work on the temporal bone to buy it, no matter what it costs, for Siebenmann called attention to the paratubal cells, which I believe are pneumatic. I believe that they are developed from a different center than the labyrinth cells.

Dr. Lewy's cisterna pontis lateralis I consider a definite entity. It has not been described because investigators have not used a proper method; viz., dyes in solution by gravity, as I have in my cadavers (*Archives of Surgery*).

In reply to Dr. Knapp in regard to the third nerve, I consider it most important. It runs alongside in the arachnoid in a lake of its own, and any trouble, no matter how slight, may cause irregularity of the pupils. The pupils should be examined carefully in every case.

The motor portion of the oculomotor nerve does not seem to be very sensitive to pressure but the sensory portion apparently responds to the slightest intervention. The sympathetic fibers that come up in the carotid and go from there to the ciliary ganglion are extremely important, as was emphasized in one of the cases reported.

I have nothing against neurologists, but as a class they are dealing with degenerative lesions while I am dealing with suppurative lesions and the same rules do not apply. My consulting neurologist takes his point of view, and believes that given a running ear and neurologic symptoms, unless the neurologic symptoms fit into something very definite they are apt to be associated with the suppuration in the ear. We see many cases with neurologic symptoms that do not go on to anything, but many do. I fully agree that a routine neurologic examination should be made in every case. In fact, there should be repeated neurologic examinations.

What I know about meningitis I have acquired from work on cadavers. In eight cadavers I injected a pigmented fluid into the ears after opening the labyrinth to find out where it would go. This showed that the arachnoid pathways recorded in the books are wrong, for in the cadavers illustrated in the books, showing the large cisterna of the base and their free communications, injections had been made under great pressure. I found that the spaces do not communicate freely with one another, but that the communications are divided by such fine trabeculae that if one opens the mastoid and the labyrinth and fills the ear full of dye, if the dye is in solution it flows from that to all parts of the arachnoid base, but if the dye is in suspension it will go a certain distance and then meet a fine space and go no further. While cerebrospinal fluid can pass, the spaces are so fine that dye in suspension cannot pass, and that is what causes a localization of the meningitis (Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, *Arch. Surg.* **20**:386 [March] 1930).

Referring to the experiment on rabbits that Dr. Grinker mentioned, I agree that the observations were correct. Chemical dye is not an irritant and does not cause adhesions but bacteria do, and for that reason the spaces are closed off, for nature makes an effort to localize all infections.

I agree concerning the danger of spinal puncture. I have seen deaths not infrequently and have learned this: If there is a suspicion of increased intracranial pressure in the posterior fossa I do not make a lumbar puncture. If there is a suspicion of an infection of the blood stream I know that I may cause a meningitis by doing a massive lumbar puncture and do not do it, although the withdrawal of a small amount for diagnosis is almost a routine.

In cases of meningitis I sometimes puncture and drain three times a day and I have never seen an accident in those cases.

Directory of Otolaryngologic and Ophthalmologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. T. E. Carmody, 227, 16th St., Denver.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Philadelphia. Time: 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 27-31, 1930.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

NEW ENGLAND OTOLOGICAL AND LARYNGOLOGICAL SOCIETY

President: Dr. Owen Smith, 690 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Stanwood Fisher, 192 State St., Portland, Maine.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Place: Victoria, B. C.. Time: Sept. 3-5, 1930.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fred Hasty, 119 Seventh Ave., Nashville, Tenn.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Louisville, Ky. Time: November, 1930.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: Dec. 10 and 11, 1930.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland, Ore.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland, Ore.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. E. Raia, 272 Broadway, Providence.
Secretary: Dr. F. W. Dimmitt, Jr., 195 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

LOCAL

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William L. McDougall, Atlanta National Bank Bldg., Atlanta.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Samuel Salinger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Clerk: Dr. Frederic M. Strouse, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John E. Brown, 370 E. Town St., Columbus, Ohio.
Secretary-Treausurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. C. C. McClelland, David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. R. J. Warner, 706 Church St., Nashville.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. William A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Watson Marshall, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Thomas B. McCollough, 121 University Pl., Pittsburgh.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Street, San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.